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ERWIN F. SMITH

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ERWIN F. SMITH

1854–1927

RODNEY H. TRUE

I suppose the purpose of a biographic sketch like this should be an attempt, while yet a man's life is vividly with us, to bring together such information concerning his efforts and achievements as have become a part of what we think and know and to recall once more those individual traits that came to be a part of the life experience of those who knew the deceased. To make such a sketch of most of us would be much simpler than to attempt to trace even the outlines of the very simple yet complex story of the years of Erwin F. Smith.

While the ground plan of his life was very simple, he developed the fundamental principle with much variation. The details were often elaborated, as his favorite Beethoven elaborated his simple themes, until a great variety seemed to appear in the pattern.

My acquaintance with our friend began at the Madison meeting of the American Association for the Advancement of Science, where I, as one of the younger men familiar with that charming region, was told off to pilot a party of botanists through the Dalles of the Wisconsin River. To my great good fortune Erwin F. Smith and Volney M. Spalding found places in my boat. As we drifted or rowed through that magic gorge, I was impressed by the close observation, eager attention, and complete appreciation shown by Dr. Smith toward everything about him, and the friendly familiarity then extended to me was never withdrawn during the succeeding years.

Thus when I was a student at Leipzig a few years later, I was not inattentive while attending Alfred Fischer's lectures in bacteriology to hear him mention an American named Smith who claimed that bacteria could grow in plants, that they did so grow and produced diseases in them as in animals. The naturally rather bitter tongue of Fischer denied this claim and laid Smith's "blunder" to a dirty technique in terms that an American present felt were intended to reflect rather broadly on the state of science in Smith's country. This was an episode in that most significant polemic

in which, like Pasteur in his day, Smith fought and fought strenuously for his glimpse of a very important truth. It gave me, who had winced as Fischer laid on the lash at Leipzig, a lively satisfaction to see Smith more than completely vindicated as work progressed.

That phase of phytopathology in which Dr. Smith gained his famous victory over Fischer and Robert Hartig was not the field of his maiden effort. Graduating from the University of Michigan in 1886, after taking a course in which biology received main emphasis, he promptly began a life of research by entering the United States Department of Agriculture in the fall of that year. He remained in this institution during the rest of his life. By some strange irony of events, better appreciated as the years have gone by, the young graduate was given the task of finding out the cause of peach yellows, a disease very destructive at that time in Delaware and Maryland. In spite of all the young investigator's enthusiasm and industry, the cause of the disease remained hidden. As he himself has written —in a summary of his work prepared in 1922 at the request of others— "After some years, I abandoned this research and devoted my time to other subjects, mainly, as I have often said, to save my reputation, but really because the problem appeared to me to be insoluble in our then state of knowledge. For that matter it has remained unsolved up to the present time."

One might speculate interestingly concerning the possible effect on Smith's later career and on the course of phytopathology in America had he attacked a less difficult problem first.

He next turned to the study of *Phytophthora infestans* and of *Plasmopara viticola*, but peach yellows, peach rosette, and later the "little peach" of Michigan and brown rot of peach were much on his mind and formed the subjects of important contributions prior to 1892.

He then turned away rather definitely from peach troubles and gave much attention from 1894 to 1910 to Fusarium injury. His work on this group of diseases on melons, cotton, cowpeas, tomatoes, potatoes, and cabbage is now a well-known chapter in American plant pathology. His paper on the "Fungous Infestation of Agricultural Soils in the United States" (1899) was the first paper on this subject published in the United States or elsewhere, insofar as it concerned Fusarium infections. This work on *Fusarium* opened the way to later work by Orton and others that resulted in the securing of highly resistant strains of most of the crop plants named.

Perhaps the greatest contribution that our country has made toward an understanding of plant disease lies in the field of bacteriology. Burrill's pioneer discovery in 1879–80 that bacteria were the cause of pear blight gave a glimpse into an unsuspected realm. It is not to be wondered at that this field, new and full of tempting obscurities, should have so powerfully

attracted the young researcher that he was able to write in 1922 that he had "done original researches on such diseases every year from that time to the present." He began his long series of brilliant and fundamental investigations with a study of cucumber wilt in 1893.

The immediately succeeding years were largely devoted to bacterial diseases. In 1897 Alfred Fischer brought out his "Vorlesungen über Bakterien," a book based on the lecture course that I had heard a few years before. In this book Fischer elaborated the attack on the work of Smith and other Americans and did it in a way that injected a rather bitter flavor into the controversy. This book brought a swift reply from Smith, who rightly felt himself called on to lead the defense. This he maintained with a thoroughness and persistence that led to the complete establishment of his claims.

I have heard Dr. Smith discuss this chapter in his scientific career in the quieter after years, and a tinge of regret always crept into his voice. That the truth should be gained by battle seemed at times still to be a necessity, something hardly to be regretted perhaps, but that the clouding of personal relations should follow was sometimes a sad price to pay. In a recent manuscript Dr. Smith refers briefly to this episode. He writes that "Fischer never forgave me, but I could not do otherwise; nor do I regret the polemic, since it cleared the air and advanced the science."

One is led to meditate a little concerning this controversy. It was intercontinental. American science had made an important discovery. Continental Europe had a habit of paying little attention to us. Pfeffer told me while I was a student at Leipzig that he did not have American botanical periodicals in the Institute, a partial file of the Botanical Gazette excepted, because there was little in them. He remarked that he never looked into them any way because he could employ his time more profitably otherwise. This was but a rather brutal way of saying truthfully that he thought little of American botany. I believe that same self-satisfied obliviousness was general. That America could originate anything new and worth while seemed to be out of the question, a possibility easily to be dismissed. When, therefore, stubborn insistence in the European periodicals, with a show of evidence, forced attention, irritation displaced obliviousness but, as scientists will eventually face facts—even those coming from an unexpected source—Smith finally won an increased measure of respect abroad for himself and for his country. It helped to break an opening through that semipermeable wall of indifference that had always more or less effectually shut out the New World.

I shall not attempt to mention and comment on his long series of contributions to our knowledge of bacterial plant diseases. He had already

written three volumes of monumental proportions on this subject before his death and had three more to write. In 1922, he wrote that he hoped to finish volume IV, the manscript of which was at that time well in hand. He never completed his great undertaking, but fortunately in 1920 he summarized much of this work in his well-known text-book, "Bacterial Diseases of Plants."

I cannot dismiss this feature of his work without mention of Dr. Smith's work on crown-gall and some of the consequences that he felt might come of it. His first acquaintance with this trouble began back in "peach-days," when in 1892–93 he sought some causal parasite for this lesion. "Bacteria," he writes, "were at that time not in mind," and as no constant features seemed to give a lead, the subject was for the time abandoned. In 1924, fresh from his work on olive tubercle, he returned to the study of overgrowths, specially that on the Paris daisy, this time with bacteria definitely in mind. He did not know till later that Cavara in Italy had already isolated a white organism from such overgrowths on grapes and had produced a few tumors with it. These crown-gall researches constituted an important item in his program after 1904. I shall not review the mass of detailed information, anatomical and otherwise, developed in this study. In his summary statement written in 1922, Dr. Smith indicated the gains made up to that point in a few sentences that may be quoted:

"We now know not only the morphology and biology of the organism causing the tumors but also that the type of the tumor varies with the part infected, that there are several strains of the organism and probably many, that isolations differ in virulence . . . that isolations are cross-inoculable to a very surprising degree . . . that some plants immune or nearly immune to certain strains respond vigorously to other strains and that some species are resistant to all strains so far as tested." He also outlines points unknown or needing further study.

In 1907, Dr. Smith conceived the idea from the involved microscopic structure of the tumor that its study might throw light on cancer, and "the more I have studied it the more analogies I have discovered," he wrote in 1922. His work on this phase of the problem brought him into active contact with others working on this scourge. That his contributions were appreciated by the medical fraternity is shown by his election to the presidency of the American Association for Cancer Research in 1925. He also received a certificate of honor in 1913 from the American Medical Association for his work on "Cancer in Plants." Many other evidences of similar tenor are not lacking.

I will not extend further this notice of his scientific work. Important matters have not been touched on, but the complete bibliography of his published work that follows will guide the reader to these.

It will also suggest the fact that Dr. Smith's interests were by no means summed up in his scientific activities.

The life work and character of Pasteur seem to have had a great fascination for him. This appears in several articles written about the great Frenchman and in numerous incidental references to him in his other work. His chief tribute, however, is seen in the translation of Emile Duclaux's "Pasteur: Histoire d'une Esprit," so appreciatively and faithfully rendered into English with the help of his assistant, Miss Florence Hedges.

I cannot close this sketch without further reference to those aspects of Dr. Smith's life that were developed outside of his laboratory. We are born with a rather complete circle of life possibilities that must inevitably be narrowed in order to give the intensity of effort demanded by our work. Thus we must early begin through neglect to kill off our possibilities one by one in order that a small part may be the more vigorous. Many of us resist more or less stoutly this forced atrophy and try to keep ourselves alive over the greatest possible part of our circumference. Others yield more readily. Dr. Smith strove with wonderful effectiveness to defend himself against the harmful results of specialization.

He developed a knowledge of French, German and Italian literature that opened to him worlds of intense pleasure. Often have I seen him pursue some theme from language to language with an enthusiasm and facility that showed how deeply he had read and thought. He read his Bible in a copy of the Vulgate; and Dante was a favorite with him, in Dante's own great language. Goethe was often quoted in the original. Seldom have I known a man, whatever his training and field of work, who brought such joy and understanding to the works of the great writers. His library was a sort of map of his mind. In it were all manner of noble things.

His ear never ceased to find delight in music more and more as the years went by, be it the music of the great poetry of the past or that poetry expressible only in mighty harmonies.

He took great delight in beautiful paintings, in sculpture, and in architecture. No road along which beauty might enter was blocked.

I think that while Dr. Smith was a true scientist to the very heart, he felt cramped by the physical world and sought greater freedom in the world of imagination where he could live as every man once in a while feels a desire to live. He returned from such escapes with sonnets or other spiritual treasure. These in part he collected and published in a limited edition that found its way to friends. In this privately printed volume, "Her Life and Mine," we see the acceptance of great sorrow and the utilization of it for the wider purposes of life.

It seems to me that Dr. Smith was fundamentally organized as artists rather than scientists are supposed to be organized. He was quick, enthusiastic, and strongly appealed to by beauty in all of its forms. I think that he may have had to learn the lesson of reserving judgment, of remaining skeptical, in short, the whole defensive attitude of science. Thus the imagination of the artist was fundamental, and by opening wide the book of nature it revealed to him the far reaches of life. Restraint and discipline made the poet into the scientist, whose great motto seemed to be to test all things, not once or twice, but many times before acceptance.

As a friend, he was quick and sympathetic, outspoken in praise and dispraise, loyal to the end.

Undoubtedly the future holds opportunities equal to those that he saw and grasped. Undoubtedly there are others who will accept them but not many can do as he did. Many of us will always miss our friend.

I am deeply indebted to Mrs. Smith and to Miss Florence Hedges, for some years associated with Dr. Smith in his laboratory and literary work, for access to material that has made possible much of what I have written. The portrait and the full bibliography that accompany his sketch are from the same source. Dr. F. V. Rand, associated for several years with Dr. Smith in his work, has also given me much appreciated help.

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The action of light on bacteria. 671–674.

The role of calcium and magnesium. 674–676.

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Detection of glukase by auxanographic methods. 752–753.

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Ustilaginoidea. 408.
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Whitney on Florida. 602.

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THE THREAD BLIGHT DISEASE CAUSED BY CORTICIUM KOLEROGA (COOKE) HÖHN., ON CITRUS AND POMACEOUS PLANTS

FREDERICK A. WOLF AND WALTER J. BACH

The name "thread blight" was first applied to a disease of tea in Northern India. It has come, however, to be employed as a designation for those diseases of various species of trees and shrubs which manifest themselves by conspicuous white to dark brown strands of fungous hyphae on the leaves, twigs, and smaller branches. Thread blights are of common occurrence in the tropics but are so rare in temperate countries as to have escaped the notice of many well-trained and experienced plant pathologists.

The attention of the staff of the United States Department of Agriculture Citrus Disease Field Laboratory, Orlando, Florida,[1] was directed in July, 1920, to a peculiar disease on grapefruits (*Citrus grandis*) and oranges (*Citrus sinensis*), which came to be known locally as "the shoe-string disease." This malady was first observed in the vicinity of Okeechobee, Fla., and had undoubtedly existed there for several years but had not previously been reported from the United States. The grove in which it was first noted is situated in a low hammock from which the towering cabbage palmettoes (*Sabal palmetto*) had not been removed prior to planting with *Citrus*, and is surrounded by a very dense forest. In due time a report (22) of the occurrence of this disease was prepared in which the causative fungus was tentatively identified as *Corticium stevensii* Burt. Positive identification has been impossible, however, until the past summer (1926), when the fungus was observed in the fruiting or basidial stage. In consequence of the finding of sporophores, a study of the morphology of the fungus was begun. It was apparent as the work progressed that the Citrus thread blight fungus is identical with hypochnose of apples and pears.

A cooperative undertaking, upon which no report has been prepared, was begun in 1923 for an investigation of hypochnose of pomaceous fruits.[2] In the light of this fact and of the results of the preliminary studies on the Citrus thread blight fungus, it was deemed advisable to modify the plans

[1] Field observations and control experiments, extending over three seasons, were made by J. R. Winston, formerly of this laboratory.

[2] It has been impossible to carry to completion this cooperative study as originally planned by Professor H. H. Whetzel, Cornell University, Ithaca, N. Y., and the senior writer. Special thanks are due Professor Whetzel, however, for his critical reading of this manuscript and for his suggestions.

so as to make a comparative study of the diseases on both Citrus and pomaceous plants and of the morphology and taxonomy of the thread blight fungi from both types of plants, and then to assemble the results in one report. The results of these studies are herein recorded.

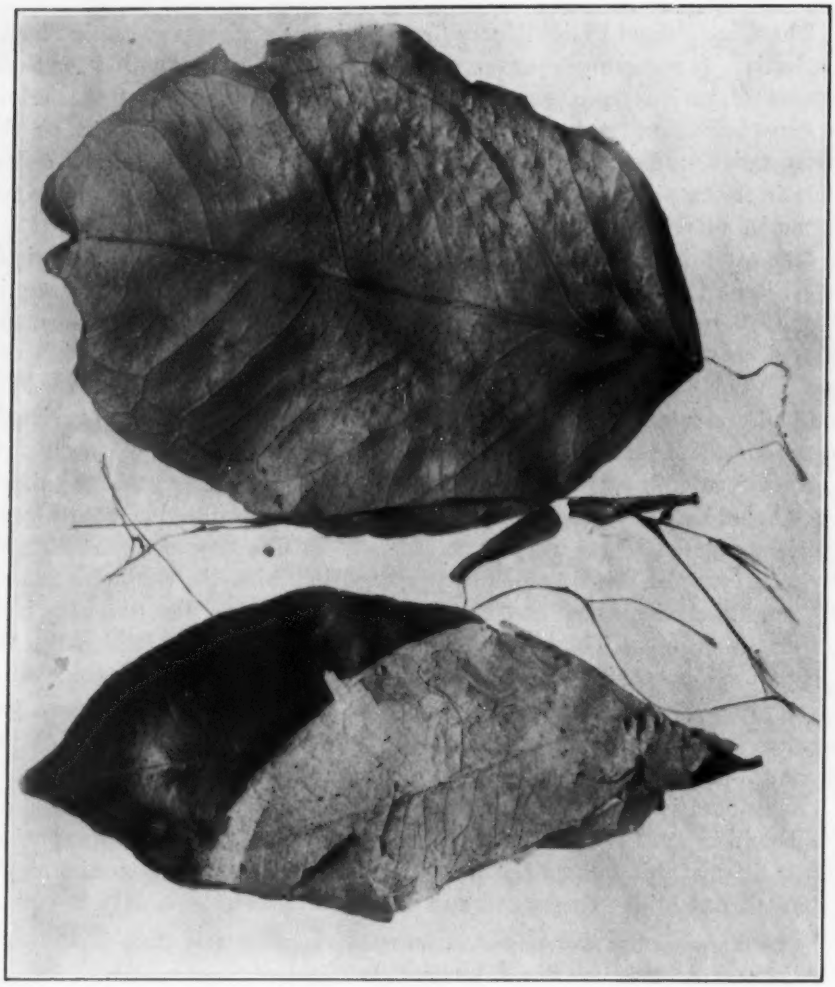

FIG. 1. Affected grapefruit leaves. The dead leaf below is attached to the green one by a hyphal mat. Rhizomorphs stripped from the twigs and attached to the petioles are shown at the center. The upper leaf bears a sporophore of *Corticium koleroga*.

HISTORY OF THE DISEASE AND HOSTS

Little attention appears to have been given by investigators to the thread blights of *Citrus*. A review of published reports indicates that at least two distinct species of *Corticium* are involved. One, *C. salmonicolor* B. and Br., mentioned by Stevenson (20), occurs not only on various species of *Citrus* and its relatives but on a wide variety of other woody plants as well. It forms pinkish sporophores upon the surface of the bark and is designated "the pink disease" throughout the tropics. The other, *C. koleroga* (Cooke) v. Höhn., is the cause of the well known "koleroga" disease of coffee (*Coffea arabica*) in Porto Rico, Jamaica, Cuba, the Lesser Antilles, Trinidad, Surinam, Venezuela, Colombia, Guatemala, Brazil, India, Java, Malaya, Queensland, and Congo, as indicated by the investigations of Stevenson (20), Cooke (4, 5, 6), Kuijper (11), Fawcett (8, 9, 10), Burt (1, 2), Stevens (18), Coleman, Venkata Rao and Narasimhan (3) and Nowell (13). The last named writer reports the presence of the same parasite on sour orange (*Citrus aurantium*), *Hibiscus, Croton, Luffa, Cucumis,* and *Codiaeum.* Additional hosts listed by Stevenson (20) include the mangosteen (*Garcinia mangostana*), the rubber tree (*Hevea brasiliensis*), and certain species of *Citrus.* Coleman and his associates (3) list as hosts in India: *Jasminum sp., Randia dumentorum, Tabernaemontana coronaria, Gardenia gummifera, Wendlandia notoniana, Canthium parviflorum,* and *Pavetta sp.*

The fungus as it occurs on pomaceous hosts in the United States has been made known through a preliminary report by Stevens (17) in 1907, which was followed, two years later, by the detailed investigations of Stevens and Hall (19). They designated it *Hypochnus ochroleucus,* a name which had been given by Noack (12) to a fungus on apple and quince which he had under observation in Brazil in 1898. Their list of hosts in North Carolina include apple, pear, quince, snowball (*Viburnum sp.*), and lilac (*Syringa vulgaris*). Burt (1 and 2) has added to this list *Codiaeum variegatum* collected in Trinidad, and Nowell (13) regards the thread blight of cacao (*Theobroma cacao*) and nutmeg (*Myristica fragrans*) in the Lesser Antilles as due to the same fungus. The same fungus,[3] to all appearances, under the name *Sclerotium pruni spinosa,* var. *ramicola,* has been collected on Mariana plum in Louisiana.

The writers' field observations on thread blight are confined to its presence on apple and pear in North Carolina and on pear, grapefruit, sweet orange, pecan (*Hicoria pecan*), and pomegranate (*Punica granatum*) in Florida. Collections[4] in the Department of Plant Pathology, University

[3] Specimens for examination were supplied from the herbarium of the New York Botanical Garden through the kindness of Dr. F. J. Seaver.

[4] These data were supplied through the courtesy of Erdman West.

of Florida, Gainesville, Fla., show that in addition it has been found in Florida to attack tung oil (*Aleurites fordii*), tallow tree (*Sapindus utilis*), persimmon (*Diospyros virginiana*), pistachio (*Pistacia chinensis*), fig (*Ficus carica*), Virginia creeper (*Ampelopsis quinquefolia*), sour orange, and rose (*Rosa sp.*).

FIG. 2. Thread blight on grapefruit showing mycelial strands, paucity of sclerotia, defoliation by breaking at the petiole and suspension of the curled, dead leaves and petioles by rhizomorphs.

OCCURRENCE OF THREAD BLIGHT ON CITRUS IN FLORIDA

The known occurrence of thread blight on *Citrus* in Florida is confined to four adjoining groves in the vicinity of Okeechobee.[5] It does not occur upon all of the trees in these groves nor is it confined to groups of trees as would be expected if the spread of infection was outward from separate foci. Instead diseased trees occur here and there throughout the grove. They can be readily detected at any time during the entire season when *Corticium* is active. This period corresponds with the rainy season which normally extends from the middle of June until the latter part of September. During the remainder of the year the fungus remains dormant and can be noticed only upon rather close inspection. Leaves, twigs, larger limbs, and fruits are involved.

APPEARANCE OF THREAD BLIGHT

On Citrus.—Attention is first directed to the disease by the presence of groups of blighted leaves promiscuously disposed throughout the tree. Those most recently killed are dry and curled and still attached. Older ones are broken off at the petiole and dangle, being suspended by mycelial threads. Closer examination shows that newly attacked leaves are still green, with whitish, powdery patches covering a portion of or the entire lower leaf surface. These patches are the fructifications of the fungus and consist of delicate, arachnoid, fungous membranes. Leaves in more advanced stages of disease exhibit large, indefinitely margined, dead areas, whose lower surfaces are covered by a brownish hyphal web. Affected leaves which are in contact may become matted together, being bound by mycelium (Fig. 1), and when they become detached from the petiole will remain dangling by brown rhizomorphs (Fig. 2).

The rhizomorphs can readily be traced from the sporophores backward along the petioles to the twigs and thence to the older wood. They course

[5] On June 28, 1927, the senior writer observed thread blight on grapefruit in a grove of approximately 100 acres located near Deep Lake, about ten miles north of Everglades, Florida. This grove is isolated by a distance of about 25 miles from the nearest planting of Citrus. The portion of the Everglades in which it is situated consists of immense swamps which surround islands of dense forest. In the section surrounding Deep Lake outcrops of coquina rock appear at the surface.

It has not been possible to determine how long the disease has been present in this grove, although in all probability it has existed there for a term of years. The information in hand as to the source of the Citrus trees and the possibility of the presence of *Corticium koleroga* on the young trees indicates that they were free from the disease when they were transplanted.

Thread blight had evidently begun to spread in the grove with the advent of the rainy season, which occurred about two weeks prior to June 28. Basidia with basidiospores were present in abundance on the newly attacked leaves in collections made on this date.

along the lower side of the twigs or on the portions least exposed to the direct rays of the sun. Chestnut brown sclerotia are sparsely present on the twigs of the current year and on those of the previous season (Fig. 2). Those on older wood are darker and manifestly desiccated and exhausted. Affected twigs die back as the result of defoliation by thread blight.

The attack on the fruit follows the extension of the mycelial strands along the fruit spurs, and the fungus appears as a conspicuous, fibrillose, anastomosing meshwork over the surface of the young fruit (Fig. 3). These strands eventually spread out and toward their extremity are separated into simple hyphal threads. The areas beneath these distal portions assume a scalded appearance owing to the death and collapse of the rind tissues (Fig. 4).

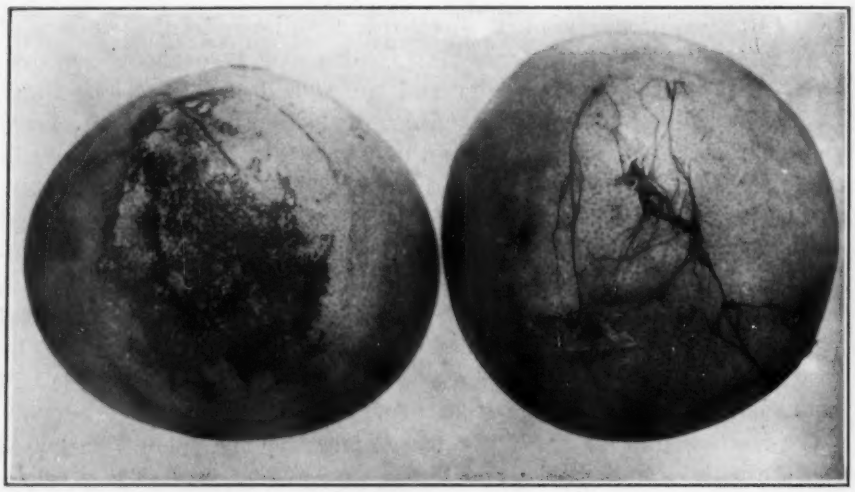

FIG. 3. Young grapefruits attacked by *Corticium koleroga*. A network of strands occur on the fruit at the right; the other one shows the initiation of the "scald" stage.

On Apple and Pear.—The writers' observations on the appearance of thread blight on apples and pears confirm in all essential features the account given by Stevens and Hall (19). At first glance affected trees simulate the appearance of fireblight in that dead leaves throughout the tree remain hanging. Closer inspection, however, of dead and dying leaves reveals the dense, brown, fungous membrane upon the lower leaf surface. This membrane is much more prominent than on *Citrus* and may be stripped off intact. On newly infected leaves which are still green this membrane

is very delicate and white. Before it has developed sufficiently to cover the entire leaf, basidia are formed in abundance, especially along the margin of the leaf (Fig. 5). These fructifications are the terminations of rhizomorphs, at first white and then brown, which extend outward along the petioles from the sclerotia on the twigs (Figs. 5 and 6). These sclerotia are hemispheric, chestnut brown bodies which are formed abundantly upon the twigs of the current year. Those on older wood are darker in color and appear dead. They may remain viable, however, until the second season after their formation. As attested by the observations of L. A. Ammon,[6] County Agent, Brevard, North Carolina, the sclerotia which were formed during 1924 remained dormant during the summer of 1925 because of the drought in western North Carolina. With the advent of abundant

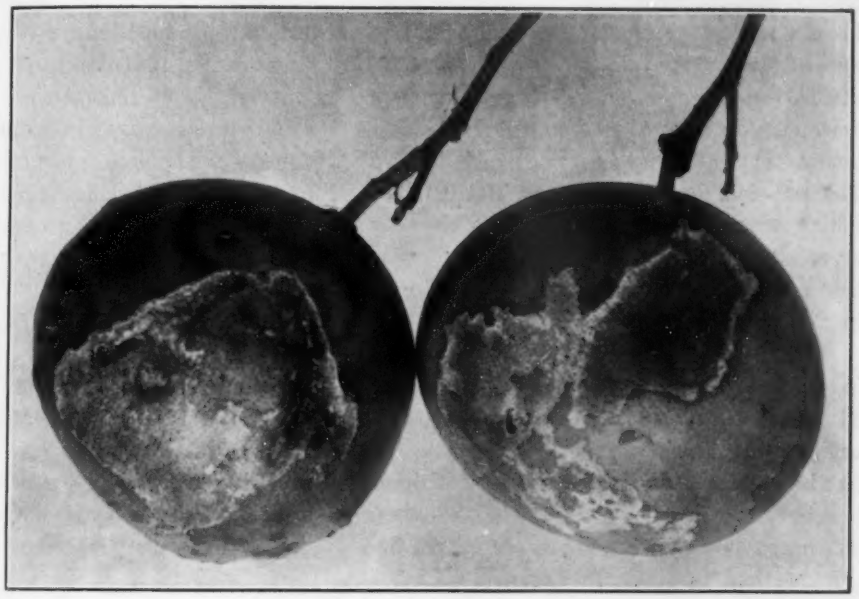

FIG. 4. Grapefruits with large "scalded" areas; mature lesions on young fruits.

rainfall late in July, 1926, dormancy was broken and the fungus spread rapidly.

The thread blight fungus does not appear to cause the death of apple and pear twigs, but trees in which the infection has persisted for years are stunted as evidenced by the abundance of short twigs.

[6] Special thanks are extended to L. A. Ammon for shipments of specimens and for the use of notes bearing on his observations on this disease.

Stevens and Hall (19) have reported that sclerotia are less frequently present on apple fruits than on twigs. This appears to be the case with all varieties observed except Russets. The entire surface of Russets may be abundantly covered with sclerotia (Fig. 7); whereas occasional fruits of other varieties bear sclerotia in the region of the blossom end or stem end only. Strands which originate from the sclerotia on twigs migrate along the fruit spurs, and thence to the fruit. Infection of the fruit follows. The organism does not appear to be the direct cause of decay of the fruits but affected fruits are rather quickly destroyed after harvesting by the common storage molds.

On Pecans.—Thread blight on pecan has been collected in the vicinity of Okeechobee, Fla., in a small group of trees about 20 years of age which are growing along the edge of a Citrus grove. The character of the disease on this host as far as size, number and distribution of sclerotia on the twigs (Fig. 8) are concerned, is entirely similar to that on apple and pear. The appearance on the foliage is somewhat different, however, in that the prominent strands which extend along the petiole from sclerotia of the previous season become very small and inconspicuous as they course outward on the leaflets and attack them progressively from the base of the leaf upward. The affected leaflets are shed in turn, beginning with the lowermost, but they may remain suspended for a time by the rhizomorphs. Membranous mycelial wefts of the consistency of those on *Citrus* are formed on the lower leaf surface. The leaf tissues involved become brown and dry, which results in the distortion of the leaves.

On Pomegranate.—The disease on pomegranate differs in appearance in several respects from that presented by pomaceous and Citrus hosts. The sclerotia on twigs are as numerous as on apples and pears, but are small, irregular in outline, and crustose (Fig. 9). The rhizomorphs commonly have ribbon-like lateral expansions. The entire lower leaf surface is covered by a very dense, tawny, fungous membrane which may be torn off in frayed strips as shown in figure 9. This membrane is considerably thicker than on any other species which have been observed.

ISOLATION OF THE FUNGUS

No account has come to hand, except that of Coleman and his coworkers (3), of the isolation and cultivation of the thread blight fungus in pure culture. Repeated unsuccessful attempts have been made to isolate it by planting sclerotia, and fragments of rhizomorphs and of mycelial wefts on agar. Either no growth at all has been secured by this method, or the growth was quickly overrun by species of *Colletotrichum* and *Pestalozzia*, which are veritable fungous weeds in Florida. Accordingly, efforts were

directed toward securing more favorable material for the isolation trials. It was found in the preliminary tests that the fungus from affected tung oil twigs could be induced to grow when placed in contact with twigs of living grapefruit trees. On June 13, tung oil twigs[7] bearing an abundance of

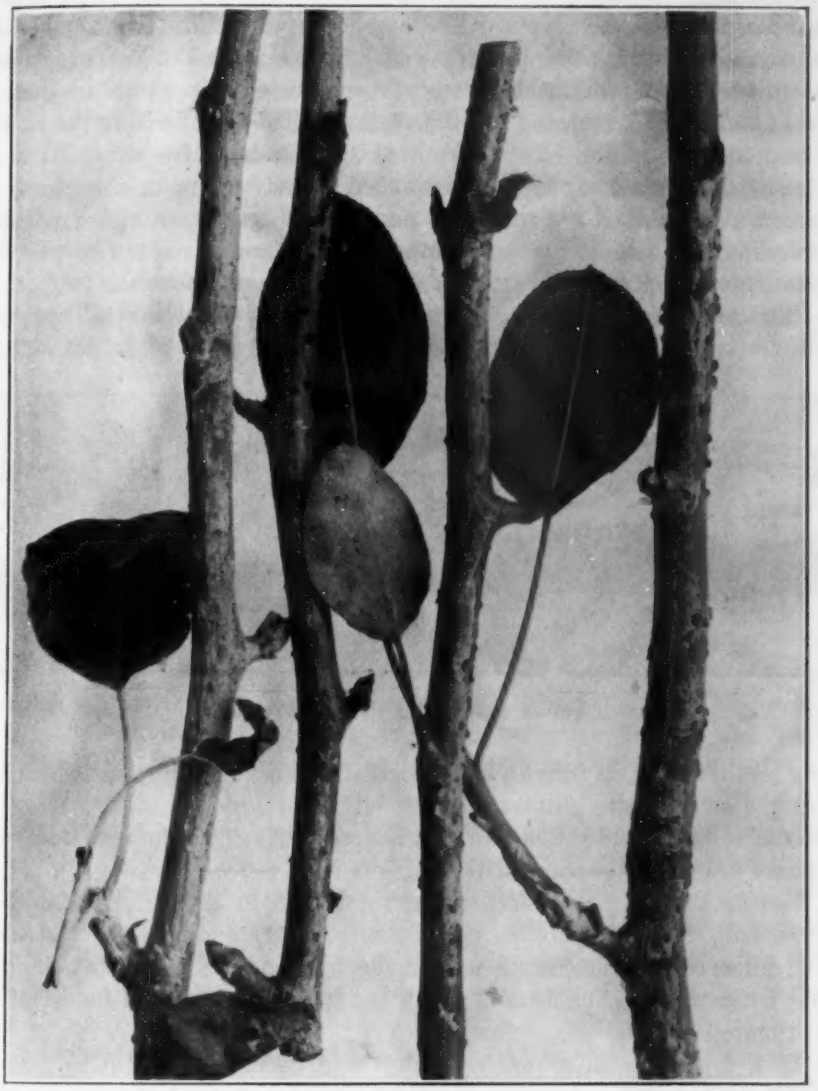

FIG. 5. Rhizomorphs, sclerotia and sporophores of *Corticium koleroga* on pear.

[7] This material was supplied through the kindness of Erdman West.

sclerotia, which had been collected April 27, were tied to young twigs of pears, grapefruits, and oranges. They were then wrapped with moist absorbent cotton and the tips of the inoculated twigs encased in paraffin paper. After seven days young rhizomorphs had developed and were found to have extended for an inch or more over the surface of all inoculated plants. Two days later, delicate white wefts had formed on the lower leaf surfaces, which upon microscopic examination proved to be sporophores. Leaves which bore fructifications were taken from each species of inoculated plant and attached, with the lower surface directed down, within the covers of poured agar plates. This permitted the basidiospores on ejection to lodge on the surface of the agar below. Within 12 hours an abundance of spores had fallen. They germinated promptly (Fig. 10), and pure cultures were obtained in these plates when reasonable care was taken to prevent contamination. Transfers were then made to various substrata by cutting out blocks of agar containing germinated basidiospores. The resultant pure cultures from this series were designated, because of their origin, as the

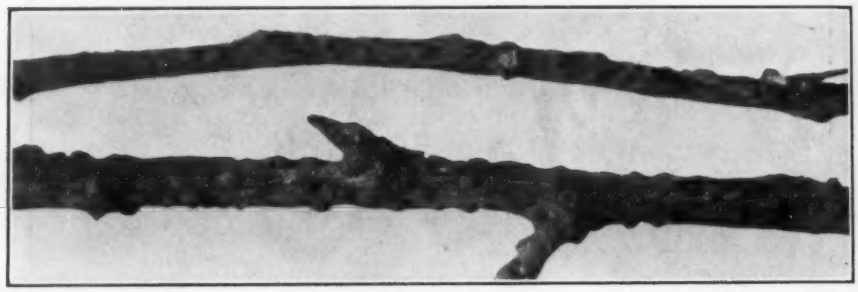

FIG. 6. Thread blight on apple twigs showing sclerotia and rhizomorphs.

tung oil thread blight fungus, a designation which was employed to identify it in the comparative cultural studies. This method of isolation is essentially like that used by Coleman and his assistants (3), who obtained pure cultures from basidiospores of this fungus from coffee.

During July, 1926, collections of thread blight in fruiting condition were made from grapefruit, pear, pomegranate, and pecan. Isolations from all except pomegranate were made by trapping the fallen basidiospores, according to the method which has just been described for isolating the tung oil fungus.

In a few cases isolations have also been effected by planting bits of recently infected leaves on agar plates. The surface of the leaves used was disinfected by immersion in alcohol; the alcohol was then removed by flaming. This method has little to commend it in isolating the thread

blight pathogene because various secondary invaders follow infection by the thread blight organism very closely and, in culture, grow out from the diseased tissues much more rapidly than the pathogene. Furthermore, the planting of old sclerotia has not proved to be a favorable means of isolating the fungus, although pure cultures have been obtained from young sclerotia.

In the comparative cultural studies use has been made of potato dextrose agar, cornmeal agar, sterilized pigeon pea stems (*Cajanus indicus*), steamed potato cylinders, and sterilized slices of immature grapefruits.

Growth on all these substrata is sufficiently rapid to form colonies of loose white mycelium within a week. They then become tinged, and at maturity are fuscous, resembling closely in color old mycelial wefts on the foliage. With the exception of a culture isolated in 1924 from sclerotia on apple twigs, no evidences of sclerotia have appeared in any of the cultures on any of these media.[8] None of these cultures have developed sporophores. Further, no discernible differences in culture have been noted between thread blight from tung oil, grapefruit, pear, and pecan.

PATHOGENICITY

The writers' first efforts to secure experimental proof of the pathogenicity of the thread blight organism were made in attempts to isolate it, as has been recounted on previous pages. Subsequently several series of crude inoculations were made. On July 1, infected twigs of pecan and of grapefruit were used to inoculate pear and grapefruit. They were applied to the twigs, wrapped with wet absorbent cotton and enclosed in paraffin paper to preserve a high relative humidity. By July 8, infections had taken place and sporophores had formed both on pear and on grapefruit from the inocula from both sources. Isolations were then made by suspending some of the infected pear and grapefruit leaves over agar plates. Pure cultures of *Corticium* were secured from basidiospores which fell and germinated on the surface of the agar.

On July 16 another series of crude inoculations on pears and grapefruits was made. In this trial, affected twigs of pear and pomegranate, which had been collected at Gainesville, Fla., a few days previously, were employed as inocula in the same manner. After eight days conspicuous sporophores had developed on each inoculated plant. By this same method of isolation pure cultures of the thread blight fungus were secured from leaves of both species artificially inoculated.

In summary, the results of the tests on pathogenicity show that infection of both pear and grapefruit readily follows the use of a crude method of inoculation of the organism from tung oil, pecan, pomegranate, pear,

[8] An abundance of sclerotia have appeared in cultures of the fungus isolated from grapefruit on June 28, 1927.

and grapefruit. It is reasonable to suppose, therefore, that reciprocal infections of any of these species would result from inoculation with the thread blight organism from any one of the others. It has not been possible to make such trials, however, because of the lack of suitable plants for use in inoculation.

Inoculations with pure cultures have been limited to the use of cultures originally isolated from tung oil, pear, and grapefruit. The inoculum was applied by cutting out bits of colonies on agar, and binding them with wet absorbent cotton on twigs of pear and grapefruit. The tips of inoculated twigs were then wrapped with paraffin paper to conserve moisture. Infections were invariably evident in the course of a week. Basidia with spores developed in due time, and after three weeks mature sclerotia had formed. The studies on pathogenicity with pure cultures of the fungus, while limited in number, confirm entirely the results of the crude inoculations.

FIG. 7. Mycelial strands and sclerotia in abundance on Russet apples.

MORPHOLOGY OF THE FUNGUS

Consideration was given to this phase of the problem to determine whether a comparison of mycelia, sclerotia, basidia, and basidiospores both from natural infections on pear, grapefruit, pecan, and pomegranate, and from artificial inoculations on pear and grapefruit would substantiate the conclusion that the thread blight disease on the various hosts is caused by one and the same fungus.

When microscopic preparations are made by mounting hyphae from fresh material of any of the plants mentioned above, they contain rather coarse elements, hyaline or slightly colored, 4.5–7 μ in diameter, which are plainly Rhizoctonia-like in method of branching and septation. No clamp

connections have been noted. The cells which comprise the sclerotia and the intercellular mycelium are binucleate (Pl. XXVI, D), a condition which is of common occurrence among Basidiomycetes. The hyphae from different sources are indistinguishable, one from the other, as shown by figure 10: A, B, and E represent hyphae from natural infection on pear, apple and grapefruit; C represents hyphae from pear artificially inoculated with the tung oil fungus, and D represents hyphae from grapefruit inoculated with the fungus from pecan. Furthermore, no discernible differences can be noted between hyphae from cultures and those from parasitized leaves and twigs.

Sclerotia from apples and grapefruits were embedded in paraffin, sectioned, and stained. These sections show that the sclerotia are always seated in clefts or crevices and never arise on smooth surfaces (Pl. XXVI, G and H). The necessity for such places of attachment for sclerotia accounts no doubt for their occurrence in abundance on the fruit of Russet apples, since their entire surface is covered with short fissures which are arranged concentrically about the stem. All other varieties of apple are known to lack these crevices or to possess them only at the blossom and stem ends.

A cross-section through a sclerotium on Russet fruits (Pl. XXVI, F) shows that it consists of brown, thin-walled cells, which typically constitute a uniform fungous parenchyma (Pl. XXVI, I). Intercellular spaces may be present, and in some the central medullary portions consist of rather loose hyphal elements (Pl. XXVI, J) such as occur in the sclerotia of *Corticium vagum* (24). There is no evidence of a differentiated cortical layer such as occurs in the sclerotia of the genus *Sclerotinia*. The outer portion is to be regarded as a pseudorind, the cells of which in mature sclerotia are more or less collapsed (24), resulting perhaps from desiccation.

The expanded basal portion which serves as an anchor or holdfast rests closely upon the pulp cells of the fruit or, as the case may be, upon the corky layer of the outer bark of the twigs. These sections show no evidence of extension of hyphae from the holdfast into the subjacent tissues. If there is any withdrawal of food material from these host tissues by the holdfast it is not manifest by a collapse of the cells. The scarcity of fissures in the bark of grapefruit twigs undoubtedly accounts for the paucity of sclerotia on this host. Those which do occur, when sectioned, as shown in Plate XXVI, B and K, are indistinguishable in structure from those on apple fruits and twigs. They are secured by holdfasts, and the tissues upon which they are closely seated appear to be normal in every respect (Pl. XXVI, K).

An examination of microscopic preparations of newly formed basidia shows that they arise as terminations of short lateral branches. They are simple, ovoid, 10–12 by 7–8 μ with four, rarely six, slender sterigmata. The

basidiospores are hyaline, flattened on the opposed faces, rounded above and tapered below, 9–13 by 3.5–5 μ, with 10.5 by 4.5 μ as the most common size. There are no detectable differences between basidia and basidiospores on the several host species as is apparent from figure 10, A and B, which represent these structures from natural infections on pear and grapefruit respectively; and C and D, which represent artificial inoculations of the tung oil fungus on pear and of the pecan fungus on grapefruit respectively. This accords entirely with the observations of Burt (1), who states that the "microscopic characters of *C. stevensii* and *C. koleroga* are within the limits of fluctuation of a single species."

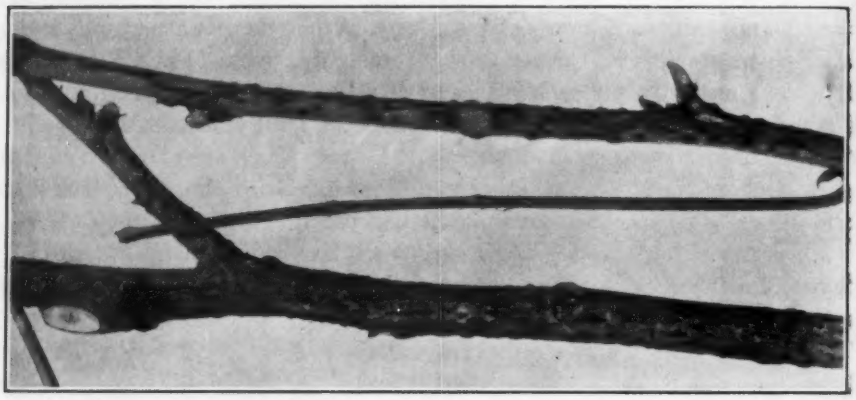

FIG. 8. Pecan twigs and petioles bearing mycelial strands and sclerotia.

PATHOLOGICAL ANATOMY

Burt (1) sectioned leaves bearing sporophores of *C. stevensii* but secured no evidence of intercellular hyphae. Neither was any evidence of penetration by *C. koleroga* found in leaves of coffee by Coleman and his coworkers (3).

Lesions on leaves of apple, pear, grapefruit, pecan, and pomegranate, and tissues of affected fruits of apple and grapefruit were sectioned in paraffin and stained with Haidenhains iron-alum haematoxylin to determine the relationship of the pathogene to the tissues. Plate XXVI, A, C, E and L, represent vertical sections of diseased leaves of grapefruit, pear, pomegranate and pecan respectively. The fungus manifestly gains entrance in all cases through the stomates and courses between the cells, involving all of the tissues.

Since no vegetative hyphae have been found in the host tissues at the base of the sclerotia, as has been indicated previously, it is entirely probable that the food necessary for sclerotial formation is taken from the leaf tissues

by the intercellular mycelium and translocated to them. At any rate the host cells of mature lesions are collapsed, and the protoplasts are shrunken and stain more deeply.

The rind tissues of diseased grapefruits are affected in the same manner as the leaf tissues. Plate XXVI, M, shows the intercellular mycelium within the tissues of the outer rind.

The present observations show the same type of relationship of pathogene to host in the case of all host species examined.

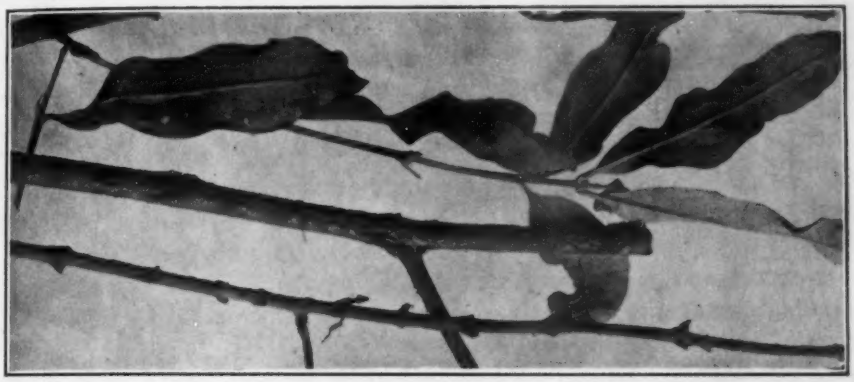

FIG. 9. Small, crustose sclerotia on pomegranate twigs with the dense, fungous membrane partially stripped off of the leaf in the upper left corner.

ETIOLOGY

The cause of thread blight is a basidiomycetous fungus, *Corticium koleroga* (Cooke) v. Höhn. This organism was first described in 1876 by Cooke (4 and 5) from collections on coffee sent from Mysore, India. He regarded it as a Hyphomycete and designated it *Pellicularia koleroga*. Two years later, Ernst (7), in his investigations of the "candelillo" disease of coffee in Venezuela, described this fungus as a powdery mildew, *Erysiphe scandens*. Fawcett (10), in 1915, compared the coffee blight fungus in Porto Rico with specimens sent from India by Dr. E. J. Butler and found them to be identical. It appears, however, from his figures of holdfast cells that he had under observation young basidia but failed to recognize the basidiomycetous nature of the pathogene.

Von Höhnel (23), in 1910, redescribed the fungus from Cocke's type and assigned to it the name *Corticium koleroga*.

The thread blight fungus on apples, pears and quinces in the southern United States was identified in 1907 by Stevens (17) as *Hypochnus ochroleucus*. Noack (12, p. 80) first noted it in Brazil as a parasite on pomaceous plants, in 1898, and named it *Hypochnopsis ochroleuca*, which was changed

in Saccardo's Sylloge Fungorum to *Hypochnus ochroleucus*. Burt (1) recognized that this fungus should properly be regarded as a *Corticium*, which differs, as he concluded, from *C. koleroga* by possessing sclerotia, and thicker, darker-colored fructifications. The supply of material upon which his observations were based was of necessity limited, however, to herbarium specimens, only two collections of which were in fruiting condition. He therefore proposed the new name *C. stevensii*, as there was already a valid *C. ochroleucum*, a binomial which had been employed by Bresadola in 1892.

The results of the present studies, which are based on the similarity of

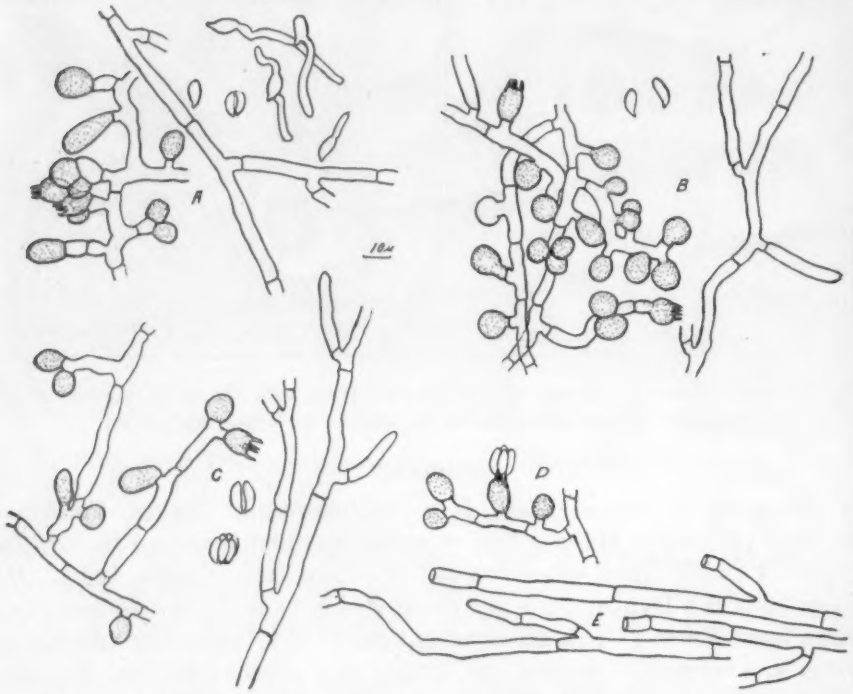

FIG. 10. A. Hyphae, basidia, basidiospores, and the germination of basidiospores of the thread blight fungus on pear, natural infection. B. Hyphae, basidia and basidiospores on grapefruit, natural infection. C. Hyphae, basidia and basidiospores of *Corticium koleroga* from pear inoculated with the tung oil fungus. D. Hyphae, basidia and basidiospores from grapefruit inoculated with the fungus from pecan. E. Mycelium of *C. koleroga* from apple fruit.

morphological and cultural characters of the organism from several host species, and on its ability to infect both pomaceous and Citrus plants, show that *C. koleroga* and *C. stevensii* are identical. The following synonomy therefore applies to this thread blight fungus.

Corticium koleroga (Cooke) v. Höhn. Sitzungsber. K. Akad. Wiss. Wien. **119**: 395. 1910.

Pellicularia koleroga Cooke, Grevillea **4**: 116. 134. 1876; Pop. Sci. Rev. **15**: 164. pl. 135. fig. A–C. 1876; Linn. Soc. Bot. Jour. **18**: 461. 1881; Sacc. Syll. Fung. **4**: 149. 1886.

Erysiphe scandens Ernst, Estudios sobre las Deformaciones, Enfermedades, y Enemigos del Arbol de Café in Venezuela, p. 16, fig. 5. 1878.

Hypochnopsis ochroleuca Noack, Boletim do Instituto Agronomico do estado de Sao Paulo em Campinas 9: 80. 1898.

Hypochnus ochroleucus Noack, Sacc. Syll. Fung. **16**: 197. 1902.

Corticium stevensii Burt. Ann. Mo. Bot. Garden **5**: 119–132. 1918.

GENERAL CONSIDERATIONS

It is a matter of considerable interest that the fungus under consideration is adapted to utilize such a wide variety of host species and is paralleled in this respect by the well-known *Corticium vagum*. The records of collections, as has been previously indicated, show that near Okeechobee, Fla., pecan, grapefruit, sweet orange and sour orange trees which are in adjacent rows are attacked; while at Gainesville, Fla., the fungus has been collected on tung oil, tallow tree, fig, pistachio, persimmon, and pomegranate, all of which were growing in close proximity. In Mysore, India, the fungus has been observed to have spread from leaves of coffee to leaves of *Jasminum sp.* in contact with them and also to have attacked various other species which were growing near (3). In the West Indies it has been collected, as previously stated, on coffee, *Citrus*, and many other species. It therefore is reasonable to assume, as the thread blight fungus from *Citrus* can attack various other plants, including Pomaceae, and the thread blight fungus from pomaceous species can infect *Citrus* and in addition several widely different species, and as the fungi from *Citrus* and pomaceous plants cannot be distinguished morphologically, that they are one and the same. The observations by various investigators on range of hosts, when interpreted in the light of the writers' infection experiments, indicate that in all probability all of the host species which have been enumerated in a previous paragraph are parasitized by one and the same species of thread blight fungus. This organism, in the light of these facts and on the basis of proper usage, should be regarded, therefore, as *Corticium koleroga*.

The most interesting points of contrast between *C. koleroga* and *C. vagum*, when it is recalled that both hibernate by means of sclerotia and that both have a Rhizoctonia vegetative stage and a basidial stage of the Corticium type, are that the former is confined to woody plants and is aerial

in habitat while the latter is confined to herbaceous plants or to seedlings which have not yet become woody, and is soil harbored.

All of the evidence in hand indicates that the sclerotia of *C. koleroga* are of primary importance in perpetuating the fungus from season to season. Once the fungus has become established on a twig or branch, it spreads by means of rhizomorphs and thus involves the new growth of each succeeding season, while at the same time the older sclerotia and rhizomorphs on the older twigs perish and are weathered away. The basidiospores which are formed during summer serve to initiate new infections on trees already diseased and, in all likelihood, are disseminated by rains, heavy dews and air currents.

This thread blight appears to be restricted to rather limited localities in any given area because of the requirement of high humidity and high temperature. This generalization is in accord with the observations of Noack (12), Stevens and Hall (19), Fawcett (9 and 10), Coleman et al. (3) and others who have studied this fungus and other closely related species. These favorable conditions obtain in the mountain valleys of North Carolina in orchards which are so situated as to be shrouded in fogs until late in the morning for days in succession. In Florida, they obtain in groves which are surrounded by heavily-wooded swamps. Such situations provide excessively high relative humidities and at the same time, because of the surrounding forest, make impossible the draining away of the heavily moisture-laden air.

CONTROL

The nature of this group of diseases indicates that the use of sprays should prove effective in their control. In consequence, various investigators have attempted, with a considerable degree of success, to control them by the use of fungicides. Fawcett (10) recommends spraying coffee in Porto Rico to check *C. koleroga* with bordeaux mixture and respraying on the reappearance of the disease. Lime sulphur sprays and sulphur dusts were ineffective in his hands. Tunstall (21) and Shaw (16), however, found that lime sulphur sprays give satisfactory control, in India, of tea diseases caused by species of *Corticium*. Each recommends, though, that the spray be applied after pruning off and destroying the infected twigs.

By the use of bordeaux mixture in combatting *C. koleroga* on coffee, Rad (14) secured a heavy crop on sprayed blocks, whereas check blocks suffered severely. Coleman and his associates (3) report that bordeaux is fairly effective in combatting the same organism on coffee when the spraying is done before the break of the monsoon.

Winston (22) has reported that *C. koleroga* on grapefruit in Florida may be successfully checked by a single application, in May, of bordeaux-oil

(3–3–50 bordeaux mixture plus 1 per cent oil as emulsion). His experiments were conducted during the three succeeding seasons 1921, 1922, and 1923. The time of application varied each year because of rainfall and temperature conditions. Spraying was done as soon as the first indication of thread blight was apparent on the foliage. As a result the disease was abruptly checked and there was no further spread throughout the rainy season, although the organism persisted in the groves, probably by means of the sclerotia. These results have been confirmed by the writers during the season of 1926. A single application of bordeaux-oil made on the advent of the rainy season effectively checked the spread of this disease, and it did not reappear during the remainder of the summer.

Quaintance (15) has suggested that spraying with bordeaux mixture should control the disease on pears. Stevens and Hall (19) have noted that it does not occur in apple orchards which are systematically sprayed. This accords with the present observations, as it has been impossible to find affected trees in orchards in North Carolina in which the disease is known to have existed formerly. During recent years, however, such orchards have been sprayed several times during the course of each season. The fact that all specimens of thread blight on apples which have come to hand were taken in unsprayed orchards is regarded as additional indirect evidence of the effectiveness of fungicides.

SUMMARY

This investigation is concerned with a thread blight disease which occurs both on citrous and pomaceous plants and also on a wide variety of other hosts. Within the United States this disease was first recorded on *Citrus* in 1920, and on apple in 1907.

The disease appears on the leaves, twigs, and fruits. It is characterized, on apples, by the presence of brown rhizomorphs and sclerotia on fruits and twigs. Affected leaves are shed and dangle, being attached by rhizomorphs. The symptoms on *Citrus* are identical with those on apple except that sclerotia are less abundantly formed.

The thread blight fungus has been isolated from basidiospores from grapefruit, pear, pecan, and tung oil and found to be indistinguishable in cultures.

Pathogenicity has been proved by reciprocal inoculations with the organism from pear and grapefruit.

No detectable morphological differences have been noted between basidia and basidiospores from the several host species. Sclerotia from apple and grapefruit are of like structure.

The organism hibernates by means of sclerotia. The disease is locally disseminated by means of basidiospores.

The infection occurs by penetration of the stomates, and the mycelium is intercellular in grapefruit, pear, pecan, and pomegranate.

The cause of the thread blight disease is *Corticium koleroga*. This identification is based upon the similarity in morphological and cultural characters of the fungus from the several host species and of its ability to infect both grapefruit and pear. *Corticium stevensii* is held to be identical with *C. koleroga*.

Observations show that high humidities and high temperatures are necessary for its spread and development.

The thread blight disease on grapefruit in Florida has been satisfactorily controlled by a single application of 3–3–50 bordeaux mixture plus 1 per cent of oil as emulsion made on the advent of the rainy season.

OFFICE OF FRUIT DISEASE INVESTIGATIONS,
BUREAU OF PLANT INDUSTRY,
UNITED STATES DEPARTMENT OF AGRICULTURE.

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EXPLANATION OF PLATE XXVI

A, C, E, F, K, L, and M are drawn to scale below A. B, D, G, H, I, and J, to scale below I.

A. Vertical section of grapefruit leaf attacked by thread blight. The mycelium occurs in the substomatal cavity and between the cells of the mesophyll and palisade parenchyma.
B. Diagram of sclerotium on grapefruit twig. The sclerotium is anchored in a cleft on the corky portion of the outer bark.
C. Vertical section of a diseased pear leaf. Manifestly infection occurs through the stomata, and the hyphae remain intercellular.
D. Binucleate cells from sclerotium and from intercellular hyphae from apple.
E. Section of diseased pomegranate leaf.
F. The basal portion, in vertical section, of a sclerotium on apple fruit, showing the relationship of the holdfast to the pulp cells.
G. Diagram of vertical section of sclerotium on apple fruit.
H. Diagram of large crustose sclerotium on apple twig, showing multiple holdfasts.
I. Compact fungous parenchyma from the basal portion of sclerotium.
J. Loose, hyphal tissue from medullary portion of sclerotium on apple.
K. Margin of the sclerotium shown in B, in detail.
L. Diseased pecan leaf in vertical section.
M. Rind of grapefruit, in section, from "scalded" area, showing intercellular hyphae and parasitized host cells.

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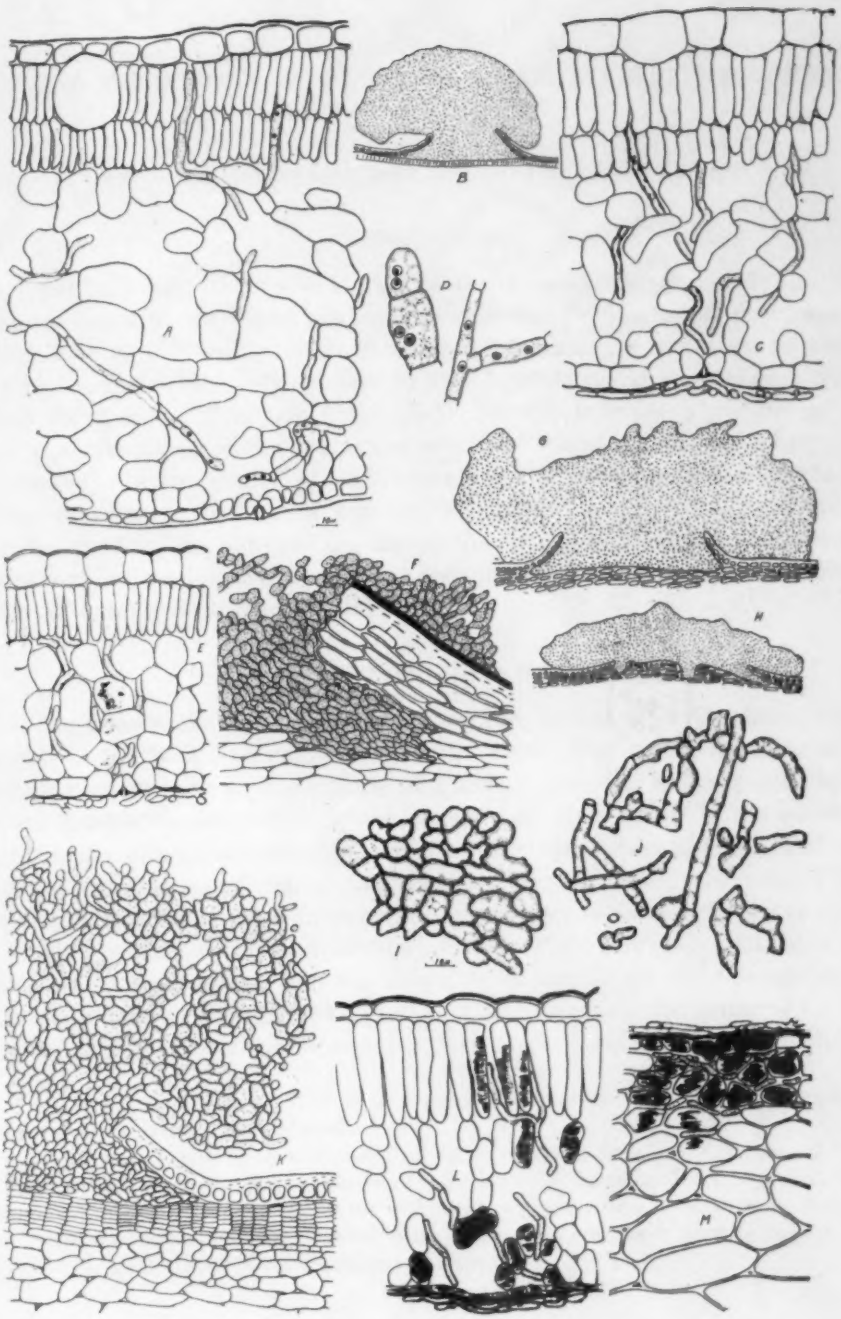
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COLOR MUTATIONS IN PUCCINIA GRAMINIS TRITICI (PERS.) ERIKSS. AND HENN.[1]

MARGARET NEWTON AND THORVALDUR JOHNSON[2]

INTRODUCTION

Although mutations are known to occur commonly among fungi, no record seems to have been made of them in the Uredinales, or rust fungi. This may be due partly to the difficulty of recognizing mutations in rust. A rust might change its spore size, or even to some degree its pathogenicity, and the change still remain undetected. Should, however, a distinct color change take place in the spore, the mutation would much more likely be observed. In the course of determining the physiologic forms of wheat stem rust present in Canada, two interesting cases of this latter type came under observation. In one case, among the normal red pustules of the stem rust an orange pustule developed, and in the other case greyish-brown pustules appeared among the red.

ORIGIN OF MUTANTS

The orange mutant had its origin in a collection from Rosthern, Saskatchewan, which was gathered in July, 1926. For six generations this rust appeared to be a pure culture of *Puccinia graminis tritici*, physiologic form 9, with the characteristic red spores of all stem rust. In the seventh generation, or approximately four months after the culture had been started in the greenhouse, an orange pustule was observed among the red. This was isolated and obtained in pure culture, and for eight months it has continued to produce nothing but orange pustules (Plate XXVII). Although so changed in color, the mutant has shown no change in infection capabilities so far as the twelve standard differential host varieties are concerned. It still has the characteristic reactions of form 9, the form from which it came.

[1] Contribution from the Division of Botany, Experimental Farms Branch, Department of Agriculture, Ottawa, Canada.

[2] The writers wish to acknowledge their indebtedness to Dr. A. T. Cameron, of the University of Manitoba, for suggestions in the chemical investigations; to Dr. C. H. Goulden for advice in determining the statistical constants; and to Mrs. D. L. Bailey for assistance in preparing the colored plate. The plate was made possible through a grant from the National Research Council of Canada.

The greyish-brown mutant[3] was found in the first generation of uredinio-spores obtained from aecia on barberries at Winnipeg in July, 1926. A few scattered greyish-brown uredinia appeared among the numerous normal red uredinia. These were successively transferred to other wheat plants until a culture was obtained in which the pustules were all of one color. As in the former case, this rust has remained constant in color, producing only greyish-brown uredinia (Plate XXVII) for over eleven months; and, as before, has shown no demonstrable change in pathogenicity from the culture in which it arose; both the normal and the abnormal strains have a reaction identical with physiologic form 36.

At first it was thought that this abnormal color might be caused by some organism parasitizing the rust spores. Repeated attempts to isolate an organism from these spores failed. Attempts were made also to transmit this condition to normal rust. The procedure was based on the theory that the supposed organism was bacterial or virus in its nature, as no fungous parasite had been observed in connection with the spores. In either case it was thought that this condition could be transmitted to normal spores by means of a filtered extract of crushed greyish-brown spores. Consequently, the latter were ground as finely as possible in a mortar, and a suspension of them in distilled water was filtered through a Büchner filter to remove the spores. The filtrate was divided into two parts, to one of which were added normal spores. A number of susceptible wheat plants were sprayed with this spore suspension. Of 81 plants sprayed, 68 became infected, but all the uredinia were of the normal color type. The other half of the filtrate was used for spraying other plants as checks, on the supposition that some viable greyish-brown spores might have passed through the filter, but no infection took place on these plants.

DESCRIPTION OF MUTANTS

The colors of the uredinia of the normal and the aberrant rust forms were classified according to Ridgway's "Color Standards and Color Nomenclature" (7). The normal rust was classified as amber brown. The color varied, however, with the age of the pustules from Sanford's brown to burnt sienna. The orange rust was classified as orange, and the greyish-brown rust corresponded to Prout's brown. For convenience, however, the original terms red, orange, and greyish-brown will be used in this discussion.

[3] During the month of June, 1927, while this paper was in preparation, inoculations from aeciospores of *Puccinia graminis* gave rise to greyish-brown uredinia similar to those described here. The physiologic form has not yet been determined. Until further information is available the term "mutant" is being used.

These differences in macroscopic appearance are accompanied by almost equally marked differences in microscopic appearance. The urediniospores of the orange rust are characterized by an entire lack of coloring-matter in the epispore, which appears hyaline after the germination of the spore. In normal rust the epispore is colored and the urediniospore retains a pale yellow color after germination. This lack of color in the spore wall of the orange spores probably accounts to a large extent for the difference in color between the two rusts. It is probable that the spore walls of the orange rust reflect more of the light than those of the red rust. The spore contents of the two rusts, on the other hand, appear similar in color. Hence, though the spores of the two rusts can be easily distinguished with the microscope where they are viewed by transmitted light, the difference is still more marked when seen by reflected light. The urediniospores of the greyish-brown rust also differ rather markedly from those of its red counterpart. The spore contents vary from grey to pale yellow in contrast with the bright orange of the normal spores. On germination they do not entirely lose their color, but remain a pale yellow like the normal spores. Great variations, however, occur in the size, shape, and color of the spores, a number of which are quite hyaline and non-viable.

FURTHER COMPARISON OF MUTANTS

Pathogenicity

As above mentioned, the infection capabilities of the abnormal rusts do not differ from those of the physiologic forms from which they mutated. It should be stated, however, that their relative pathogenicity has been tested only to the twelve differential hosts used in determining physiologic forms of *Puccinia graminis tritici* (11). No further comparison in infection type has been made between the normal rust and the mutants. Hence, as far as infection capability is concerned, the orange rust does not differ from the normal physiologic form 9, and similarly the greyish-brown rust answers to the infection type for form 36. The normal red rust produces spores in slightly greater abundance than do the two mutants, but this difference is not very appreciable and does not form a justifiable basis for distinguishing these from the red forms.

Spore Germination

A comparison was made of the viability of fresh urediniospores of the normal rust with that of the two mutants. The urediniospores were produced in the greenhouse on the thoroughly susceptible variety Little Club, and were tested for germination when the uredinia were about two weeks old.

TABLE 1.—*Germination of normal, orange, and greyish-brown urediniospores of Puccinia graminis tritici*

Normal				Orange			Greyish-brown		
Date of test, 1927	Total no. of spores	No. spores germinated	Percentage	Total no. of spores	No. spores germinated	Percentage	Total no. of spores	No. spores germinated	Percentage
Jan. 8	306	289	94.44	456	109	23.90	620	342	55.16
do 11	372	335	90.05	732	402	54.92	633	290	45.82
do 15	238	197	82.77	562	365	64.95	.....	.....	.....
do 19	500	332	66.40	1025	618	60.29	354	142	40.11
do 19	156	137	87.83	583	344	59.00	331	125	37.76
do 20	235	195	82.98	1039	738	71.03	846	367	43.38
do 25	.....	.....	.....	.....	.....	.....	505	299	59.21
do 26	368	322	87.50	934	550	58.89	725	434	59.86
do 28	369	332	89.97	604	461	76.33	641	373	58.19
do 31	423	378	89.36	792	702	88.64	459	293	63.84
Feb. 2	.....	.....	.....	433	259	59.82	432	294	68.06
do 4	333	306	91.89	717	401	55.93	410	219	53.41
			Mean = 86.32			Mean = 61.24			Mean = 53.16
				$M_N = 86.32$ $M_O = 61.24$ $M_{N-O} = 24.93$ $\sigma_{N-O} = 9.9182$ $t = 7.53$ Odds enormous			$M_N = 86.32$ $M_G = 53.16$ $M_{N-G} = 35.88$ $\sigma_{N-G} = 12.25$ $t = 9.27$ Odds enormous		

Some preliminary experiments were made to determine the best method for germination, tap water and distilled water being used in Syracuse watch glasses and in hanging-drops in Van Tieghem cells. Tap water was found preferable to distilled water, but little difference was observable between germination in the watch glasses and the hanging-drops, provided the latter were frequently aerated. The method finally adopted was to germinate spores in hanging-drops in the Van Tieghem cells for 48 hours, during which time the cells were lifted off the glass slide several times for aeration. The chief advantage in the use of the cells lies in the greater ease and accuracy with which the count can be made.

Table 1 represents the germination of the normal red, the orange, and the greyish-brown urediniospores. Each of the mutants, when compared with normal rust, shows a highly significant difference in the percentage of germination. The odds of significance were calculated from "Student's" tables of the distribution of t (12). Not only is the germination of the spores of both mutants considerably lower than that of the normal rust spores, but there is also a greater variation between consecutive tests than occurs in normal spores. The reason for this fluctuation is not clear, although it is possibly due to a more delicate response to adverse environmental conditions than exists in the normal spores. As the spores of both mutants are less viable than the normal spores, these fluctuations are not surprising.

Statistical Studies of Urediniospore Sizes

Methods of spore measurement.—The spores were projected through a microscope on to a paper screen. The magnification was standardized by means of a slide micrometer so that one small division (1/100 mm.) or 10 μ on the micrometer corresponded to one centimeter on the screen. Therefore one micron on the slide micrometer was represented by 1000 μ on the screen. By means of calipers and a steel rule the measurements of the spores were obtained in microns at a magnification of 1000.

In preliminary experiments to determine what represented accurate random sampling of the urediniospores for length and width, it was found that considerable variation occurred among samples of 100 spores when taken from single pustules of the same host. Increasing the size of these samples did not overcome the difficulty, but when four or five of the small samples from the different pustules were combined, uniformity was obtained in curve type as well as in the magnitude of the calculated statistical constants.

According to Levine (3), urediniospores of a single physiologic form from thoroughly susceptible hosts do not differ in type even if the hosts are

different varieties, although spores of the same form from a resistant variety are markedly different in type. Consequently, in this study, the spores were collected only from susceptible hosts, the variety Little Club being used throughout. All the plants were grown under identical conditions, and uredinia of the same age were used as a source of spore material. Populations of 500 spores were thought sufficiently large. Each of these populations was composed of five random samples of 100 spores each. The spores were mounted in glycerine and measured immediately after mounting.

Spore sizes of form 9, red and orange.—Two series of measurements were made of the red and orange rusts. The first series of measurements, represented by figure 1, was made in January when light conditions were

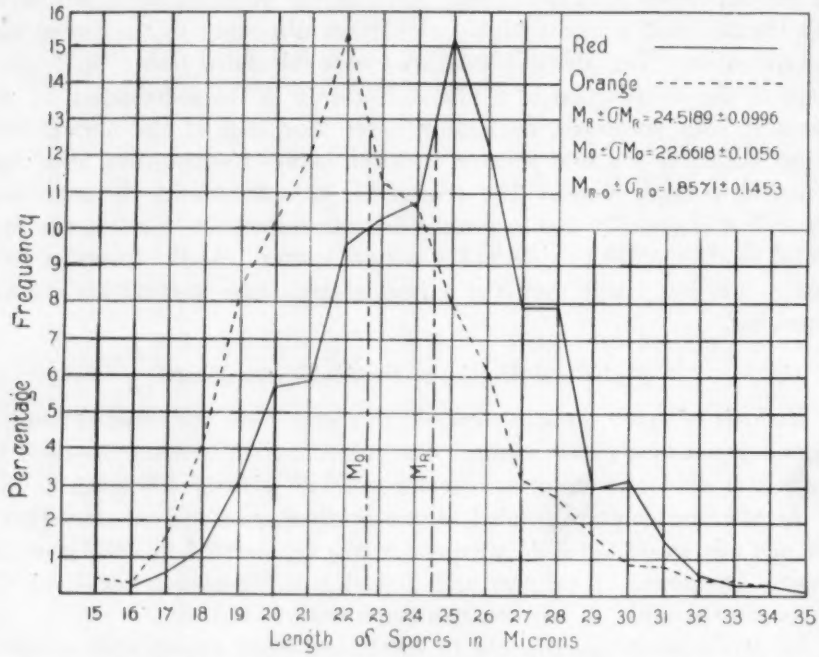

FIG. 1. Percentage frequency distributions for length of 979 spores of form 9 red (normal), and 887 spores of form 9 orange (mutant).

rather poor for rust development. The second series of measurements, as shown in figure 2, was made more than two months later, in April, when light conditions were almost ideal for the development of host and fungus. Both length and width were measured, but as the variations in width did not appear significant, the graphs are concerned only with the length.

The frequency distributions for length and width of spores are given in table 2.

Figure 1 shows plainly a significant difference between the lengths of the red and orange spores. The measurements of the widths of the spores also seem to show a significant difference. The mean width of the red spores in microns is 14.2520 ± 0.0669, that of the orange spores 14.7099 ± 0.0604. The difference between the means is 0.4579 ± 0.0280.

TABLE 2.—*Frequency distributions for length and width of urediniospores of form 9 red and orange. First series of measurements*

Length of spores			Width of spores		
Microns	Form 9 red	Form 9 orange	Microns	Form 9 red	Form 9 orange
15	.....	4	8	1	.....
16	2	3	9	5	.....
17	6	14	10	30	5
18	13	38	11	69	27
19	31	75	12	119	64
20	56	91	13	140	131
21	58	108	14	142	167
22	94	137	15	175	205
23	101	102	16	154	155
24	106	94	17	101	74
25	149	71	18	43	41
26	120	55	19	4	14
27	77	29	20	1	3
28	77	25			
29	29	15			
30	32	8			
31	15	7			
32	6	4			
33	3	4			
34	3	3			
35	1	.....			
Totals	979	887		984	886

Figure 2, as stated above, represents the second series of measurements of the spores of the red and orange rusts made two months later under more favorable conditions for spore development. This shows an even more significant difference between the lengths of the spores of the normal rust and of the mutant than the first series. It will be noticed that the mean lengths, in the case of both rusts, are somewhat greater than in the earlier measurements. This may be attributed probably to more vigorous spore development under conditions of longer daylight and more congenial temperature. The mean width in microns is 14.6934 ± 0.0868 for the red

spores, and 14.3300 ± 0.0738 for the orange. The difference between the means is 0.3634 ± 0.1139. This difference should probably not be considered significant, especially as it is directly opposite to that secured in the first measurement of the spore width of these two rusts. In general, although the measurements for spore width are reported, the writers prefer to draw conclusions only from the more obviously significant differences in spore length.

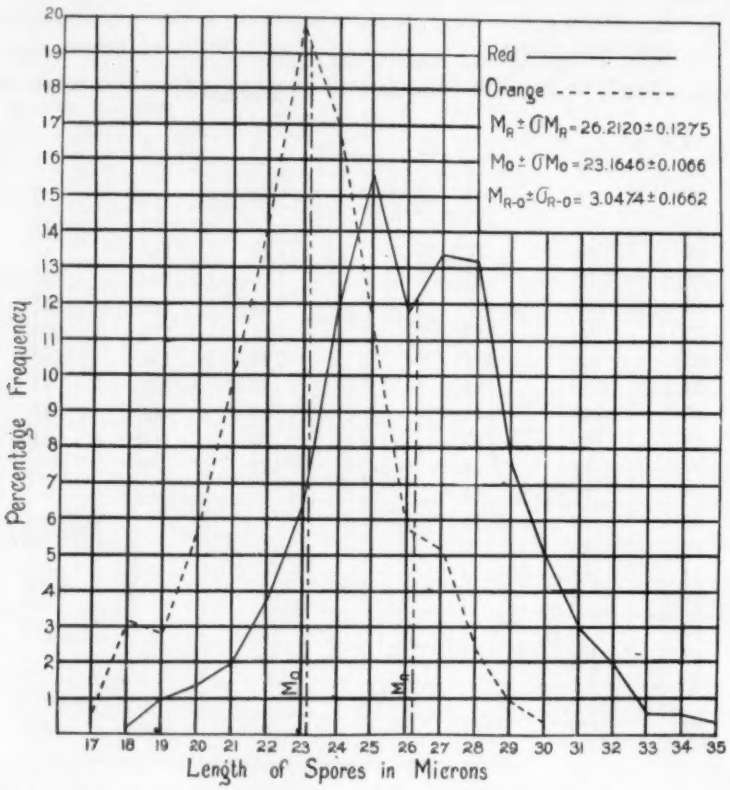

FIG. 2. Percentage frequency distributions for length of 500 spores of form 9 red (normal) and 498 spores of form 9 orange (mutant). Second series of measurements.

The frequency distributions for spore length and width as obtained in the second set of measurements are given in table 3.

Spore sizes of form 36, red and greyish-brown.—Figure 3 represents measurements of 500 spores of the normal form 36 and an equal number of spores of its greyish-brown mutant. The figure shows a significant difference in spore length. Both measurements are represented by a normal type

of curve, although that of the mutant shows a greater range than that of the normal rust form. This is due to the greater variation in length and shape of the spores of the mutant, and these variations are quite noticeable on examination with the microscope. The measurements of the widths of the spores reveal a difference which is, however, of doubtful significance. The mean width of the red spores in microns is 15.1142 ± 0.0843, that of the greyish-brown 14.7120 ± 0.1086. The difference between the means is 0.4022 ± 0.1375.

TABLE 3.—*Frequency distribution for length and width of urediniospores of form 9 red and orange. Second series of measurements*

Length of spores			Width of spores		
Microns	Form 9 red	Form 9 orange	Microns	Form 9 red	Form 9 orange
17	—	3	9	—	1
18	1	16	10	4	1
19	5	14	11	17	18
20	7	29	12	47	53
21	10	50	13	79	85
22	19	71	14	82	99
23	32	99	15	105	126
24	60	85	16	77	72
25	78	57	17	53	31
26	59	29	18	21	14
27	67	26	19	11	—
28	66	12	20	3	
29	38	5			
30	25	2			
31	15				
32	10				
33	3				
34	3				
35	2				
Totals	500	498		499	500

The frequency distributions for length and width of the spores of the normal red and the abnormal greyish-brown rust are given in table 4.

Nuclear condition of mutants.—Dodge (1), working with *Caeoma nitens*, the orange rust on *Rubus*, was able to show that in two of the short-cycled rusts which he investigated a definite color of the aecium was often associated with certain nuclear phenomena and with certain spore sizes. Usually in the reddish-orange sori there occurred chiefly binucleated spores; while in the yellowish-orange aecia, chiefly uninucleated spores. Also, when

the aecia were yellowish-orange as contrasted with those of reddish-orange color, the spores were found to be smaller and less uniform in size and shape.

Although Dodge was dealing with a different rust and a different stage in the cycle of the rust from that studied by the writers, it was thought advisable, owing to similar differences in spore size and color, to make a cytological study of the urediniospores of the normal rust and of the two mutants.

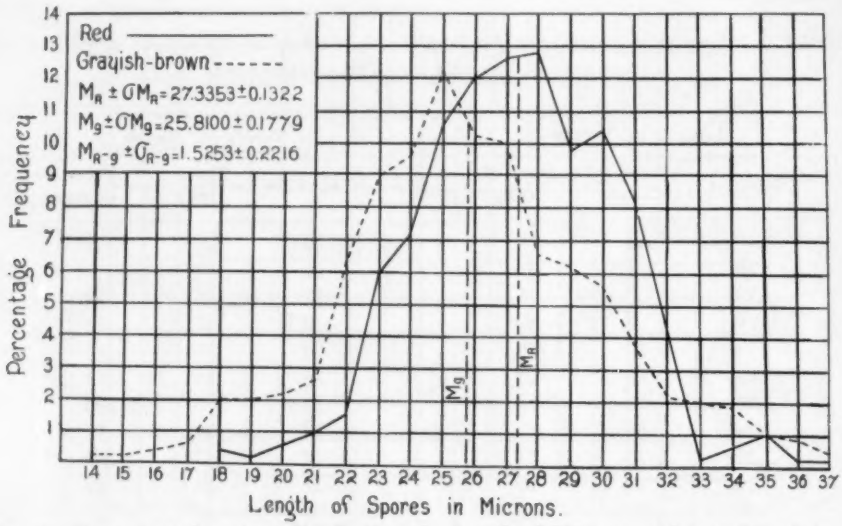

FIG. 3. Percentage frequency distributions for length of 500 spores of form 36 red (normal), and 500 spores of form 36 greyish-brown (mutant).

Little Club, a wheat susceptible both to forms 9 and 36 as well as to both mutants, was used for all infections. When the pustules were just breaking through the epidermis of the wheat plants, the leaves were embedded in paraffin in the usual manner, and sections cut from 5 to 10 μ thick. Iron-alum haematoxylin was the chief stain used.

A study of a large number of slides revealed nothing unusual in the nuclear condition of the urediniospores of either the orange or the greyish-brown mutant. In the abnormal as well as in the normal rust, all the younger spores were distinctly binucleated (Fig. 4).

PRELIMINARY CHEMICAL INVESTIGATION OF THE COLORED COMPOUNDS IN THE NORMAL RUST AND IN THE MUTANTS

The appearance of the spores of these rusts suggests the presence of carotin or xanthophyll or both, lycopin or rhodoxanthin being also possibilities. The following tests have therefore been carried out:

TABLE 4.—*Frequency distributions for length and width of urediniospores of form 36 red and greyish-brown*

Length of spores			Width of spores		
Microns	Form 36 red	Form 36 greyish-brown	Microns	Form 36 red	Form 36 greyish-brown
14		1	8		1
15		1	9		2
16		2	10	1	18
17		3	11	13	27
18	2	10	12	37	50
19	1	10	13	58	64
20	3	16	14	68	65
21	5	18	15	96	87
22	8	32	16	101	64
23	30	45	17	76	60
24	36	48	18	41	30
25	53	61	19	7	20
26	60	51	20	1	10
27	63	50	21	—	2
28	64	33			
29	49	31			
30	54	28			
31	41	19			
32	21	11			
33	1	10			
34	3	9			
35	5	5			
36	1	4			
37	1	2			
Totals	500	500		499	500

(a) Extraction with carbon bisulphide gave orange-colored solutions with the normal rust and the orange mutant, but no colored material was extracted from the greyish-brown rust. Using equal weights of the powdered rust material of the first two and equal volumes of carbon bisulphide, solutions of almost the same intensity were obtained, and these examined in a Hilger constant deviation spectrometer showed a band in the correct position for carotin, the red end being in two series of experiments, respectively 537 and 537.5 μμ for the normal rust, and 536 and 536 μμ for the orange. The other end could not be determined accurately. No bands were observed in the solution from the greyish-brown rust. Willstätter and Stoll (13) state that the corresponding carotin band has the limits 524–510 μμ in carbon bisulphide. This result suggests that carotin may be pres-

ent to an equal extent in both the normal rust and the orange mutant, but is certainly absent from the greyish-brown mutant. [The spectrometer had too wide a dispersion to distinguish the band in the blue—*Vid.* Palmer (6), p. 221.]

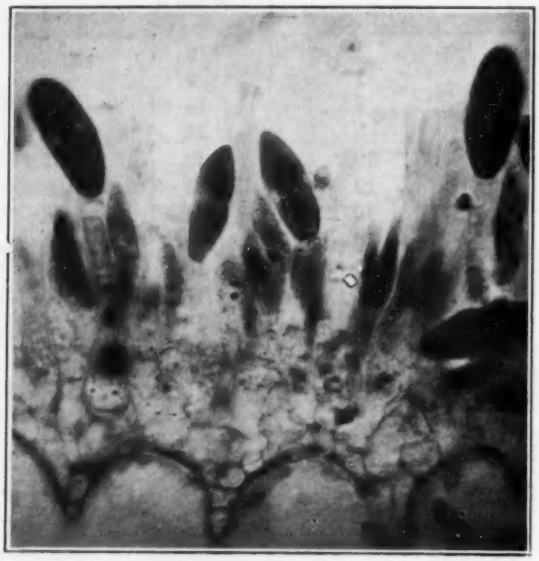

FIG. 4.—Section through a uredinium of the greyish-brown mutant × 700. (Photomicrograph by Dr. A. Savage.)

(b) Eighty per cent methyl alcohol extracted no color from any of the three. According to Jörgensen and Stiles (2), xanthophyll, if present, should be extracted. Xanthophyll therefore appears to be absent from these rusts, and this result shows further that the band seen in the spectrometer was not due to xanthophyll, strengthening the evidence in favor of carotin.

(c) Alcohol or acetone should give a rose pink extract if rhodoxanthin is present. No such color has been observed in alcoholic or acetone solutions from any of these rusts.

(d) The position of the band observed definitely excludes lycopin from the normal and the orange rust, and the absence of extractable material with carbon bisulphide also excludes it from the greyish-brown rust.

(e) Addition of concentrated sulphuric acid to powdered normal rust gave a slight blue color, while the orange mutant gave a much more intense blue, and the greyish-brown no trace of blue color. According to Palmer (6) this test is given by both carotin and xanthophyll. The difference of

intensity obtained under comparable conditions with the first two rusts, while confirming the probable presence of carotin, suggests that some additional reacting substance may be present in the orange mutant.

(f) Monteverde and Lubimenko (4) state as a specific test for xanthophyll that a green color is produced on the addition of formic acid, but, as Palmer (6) points out, this test has been mentioned only by these investigators. The writers found that the orange rust gave a very slight positive reaction, the normal rust a somewhat stronger reaction, and the greyish-brown a negative reaction, while dandelion heads employed as source of xanthophyll (6, 10) gave a much more intense color. In the absence of further evidence as to the specificity of this reaction, the writers doubt if definite conclusions can be drawn from their results with it.

(g) The powdered greyish-brown mutant has been treated with other organic fat solvents—acetone, ether, chloroform, alcohol, etc., and with boiling water, and boiling dilute acetic acid. The red coloring material was not extracted by any of these reagents. Similar treatments of the powdered red spores after the orange coloring matter had been removed by carbon bisulphide failed to extract the red. Both carbon bisulphide and acetone removed all the color from the orange rust, leaving it a chalky white. It seems, therefore, that the parent normal rust has at least two distinct color substances, orange and red, while neither of the mutants possesses both. The normal rust and the orange mutant have orange in common, apparently located in the cytoplasm of the spore. The normal rust and the greyish-brown mutant have red in common, apparently located in the spore wall and insoluble in any of the reagents used.

Conclusions. The above evidence suggests strongly that the normal rust and the orange mutant contain carotin, this being responsible for the orange color. On the other hand, since equal weights of these two rusts extracted by equal amounts of carbon bisulphide gave colored solutions of equal intensity with a corresponding absorption band, it would appear that carotin can not be responsible for the prevailing color of the normal rust. As it was not possible to extract the red color from either the normal rust or the greyish-brown mutant by the treatments employed, it seems possible that there is present in both of these some unknown red compound which is markedly resistant to solvents, which is neither carotin, xanthophyll, lycopin, nor rhodoxanthin, and indeed is not a carotinoid. The authors are continuing work to endeavor to ascertain something of the nature of this coloring matter.

SUMMARY

1. Two color mutations, one of which is bright orange and the other greyish-brown, have been observed in the uredinial stage of *Puccinia grami-*

nis tritici. The aecial and teliai stages of the mutants have not yet been studied.

2. The orange mutant appeared as a single pustule in a culture which for six generations of uredinia had produced spores of normal color; the greyish-brown mutant appeared in the first uredinial generation of a culture derived from aecia on *Berberis vulgaris.*

3. Although so changed in color, the mutants have shown no change in infection capabilities so far as the twelve standard differential host varieties are concerned. The orange mutant still has the characteristic reaction of physiologic form 9, from which it arose; and, similarly, the greyish-brown mutant remains identical pathogenically with form 36.

4. Both mutants differ markedly from the normal rust in the viability of their urediniospores. The average germination of the normal urediniospores was 86.32 per cent, of the orange mutant 61.24 per cent, and of the greyish-brown 53.16 per cent.

5. There is a significant difference in size between the urediniospores of the normal rust and those of the mutants, the former being markedly longer, but of approximately the same width.

6. The urediniospores of both mutants are binucleate.

7. Microscopic examination shows that the spore walls of the orange rust are colorless, while in the greyish-brown and in the normal rust they are colored. On the other hand, the cytoplasm of the orange and the normal spores is colored, while that of the greyish-brown spores is practically colorless.

A chemical investigation adduced strong evidence that carotin was present in the normal and in the orange spores, but not in the greyish-brown spores. The reddish-brown color in the normal and the greyish-brown spores appears to be due to some unidentified compound which is not a carotinoid, although of course the identity of the two reddish-brown colors can as yet only be assumed.

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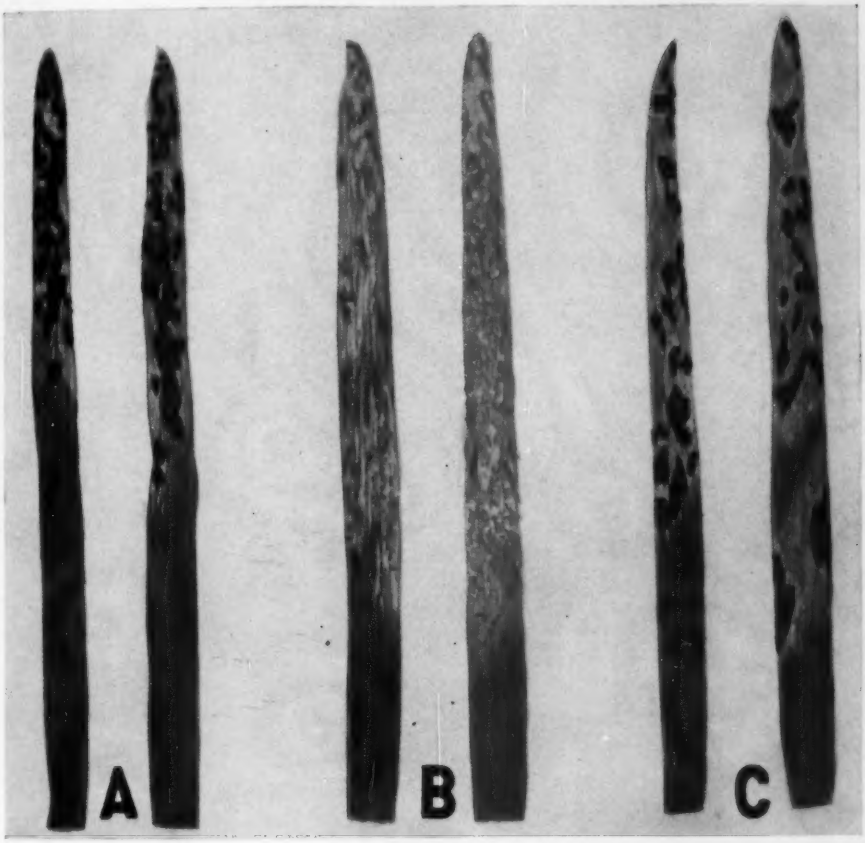
EXPLANATION OF COLORED PLATE XXVII

Leaves of Little Club wheat inoculated with *Puccinia graminis tritici:*

A. Normal rust.
B. Orange mutant.
C. Greyish-brown mutant.



Most of the striking differences in color between A (normal rust) and C (grayish-brown mutant) have been lost in reproduction. See "Description of Mutants" in text for exact colors.

UNIV. OF MICH.

TOBACCO MOSAIC ON POTATOES

F. M. Blodgett

In several recently published papers there are some seeming discrepancies in the results secured by inoculating potatoes with the tobacco mosaic virus. Johnson (6) states that "Tobacco mosaic produces brown or black necrotic lesions on the stems and petioles of potatoes apparently at the points of inoculations. Tobacco mosaic infection was not found to be systemic in potato, however, and it cannot therefore be said to be a typical host of tobacco mosaic." On the other hand, Fernow (5) reports symptoms that closely parallel those of streak of potatoes as described by Schultz and Folsom. "The petioles and the young stems become affected with a severe necrotic streaking, and affected shoots generally die soon after. . . . The second generation plants are usually much dwarfed and the leaves may show from one to five yellowish spots which become brown. . . . The petioles and stems are often affected with a more severe necrotic streaking than in the first generation plants, and the older leaves are dropped, leaving only a small crown." In this case the virus is obviously systemic, as the entire plant is affected, and the virus is carried over by the tubers to the second generation. Quanjer (8) concluded that tobacco mosaic is identical with or at least closely related to potato mosaic. This conclusion seems to have been reached as the result of a transfer of mosaic from tomato to one variety of potato by grafting, but it is not clear in one paper (8, p. 42) whether the mosaic transferred was tobacco mosaic. In a paper by Quanjer (9, p. 26) where reference is made to the same or another experiment of a similar kind, potato mosaic seems to have been the one transferred, although it is perhaps equally likely that the disease transferred was stipple-streak. This suggests itself as Quanjer reports obtaining this transfer only from the variety Zeeuwsche Blauwe and a failure from the varieties Eigenheimer and Bravo. Of the variety Zeeuwsche Blauwe, Atanasoff (2) says: "the variety as far as studied by the writer and perhaps in its entirety is infected with stipple-streak and always carries its virus, although under natural conditions it never shows the slightest symptoms of disease." Clinton (3) reports one positive case of infection with tobacco mosaic on a potato seedling. A number of unsuccessful attempts to inoculate potatoes with tobacco mosaic virus have been reported in literature by Allard (1), Dickson (4), Clinton (3), and Schultz and Folsom (10).

Apparently rather puzzling is the fact that Fernow (5) and Johnson (6) report quite different results from inoculating potatoes with tobacco mosaic virus, as there seems to be good reason to believe the work was well done in both cases and sufficient numbers used to give reliable results. Some experiments performed by the writer in 1920, and not previously reported, have a bearing on this discrepancy. In January, 1920, there were growing in the greenhouse eight tuber units of potatoes of the Green Mountain type selected from a relatively healthy commercial field. These were numbered 50 to 57, and the pieces from each potato labelled A, B, C, D. On January 21, tomato cions affected with tobacco mosaic were grafted on plants 52C, 53C, and 56C. The inoculated plants thus belonged to different tuber units. The plants lettered A and the remainder of the plants lettered C were treated in various other ways which need not be detailed here. The B and D plants of each tuber unit were left as untreated checks. All of these plants were in small pots, made little further growth, and no symptoms of infection were noticed in any of them. All plants were dug in April and planted again on July 13 of the same year. The tuber progeny of each plant of the previous generation was kept separate. Altogether there were in the series 82 plants which were tuber progeny of 30 plants in the previous series. Six of these were from the grafted plants, 52C, 53C, and 56C. Eleven plants were from the uninoculated B and D plants in the same tuber units, 52, 53, and 56, and thus served as checks on the original condition of these units with respect to freedom from mosaic diseases. All of these plants, except the 6 plants from 52C, 53C, and 56C, were large and healthy and grew vigorously. The six plants from the graft-inoculated plants were strikingly different. They were minute dwarfs, never exceeding about 5 inches in height. The leaves started to unfold, but instead of completing the process, they turned yellow and dropped soon after. The resultant plant was small, bare stemmed, with streaking on stems and petioles, and with a small tuft of partly unfolded leaves at the top. Thus these results seem to agree with those reported by Fernow, in that the inoculation of potatoes with tobacco mosaic virus produced a systemic invasion, the virus being carried over through the tubers, and the symptoms in the tuber progeny resembling those of streak of potatoes. It should be noted that these plants were grown in the greenhouse in the summer time so that rather high temperatures prevailed and thus, as will be seen later, symptoms typical of high temperature conditions appeared.

In the spring of 1926, after the papers by Johnson and Fernow had been published, and the question of the seeming discrepancy of results was raised, further inoculation experiments were undertaken with the hope of clearing up the matter. To secure tobacco mosaic, a mosaic tobacco extract

bottled in August, 1920, was used to inoculate tobacco plants. It was believed that this extract, because of its age and the known longevity of the tobacco mosaic virus as compared with certain other viruses, would be as certain a source of the unmixed virus of tobacco mosaic as could be readily obtained. The inoculated tobacco plants developed typical tobacco mosaic.

The method of inoculation from mosaic tobacco to potatoes was essentialiy the same as that described by Johnson (6). Mosaic tobacco leaves were crushed in a small amount of water with a steam sterilized mortar and pestle. The inoculations were made with a flame sterilized needle on which a small wad of absorbent cotton was wound near the tip. Sterile forceps were used to hold the stems or leaves of the plant to be inoculated where this was required. This method has been used rather extensively in other experiments. It seems to be an effective way of making inoculations with a number of viruses and introduces very little danger of contamination.

In this manner five Bliss Triumph potato plants were inoculated in the greenhouse with the tobacco mosaic virus from tobacco on April 2, 1926. The plants were 6 to 8 inches high, grown for indexing purposes under conditions favorable for the development of mosaic symptoms, and had been selected as healthy. They were transplanted to large pots before inoculation so conditions would be favorable for continued growth. At the same time inoculations were made with five other viruses on other Bliss Triumph potatoes, and five check plants were punctured with the same needle sterilized in the usual way. Numerous other plants of the same lot remained uninoculated. Inoculations were made by puncturing the plants rather freely, ten punctures in each of several lower nodes and in each of three leaflets. Within a week, necrotic lesions were noted around the points where inoculations were made with tobacco mosaic; and within a month some of the lower leaves had turned yellow and fallen. There did not, however, seem to be any indication of systemic infection at any time. The lower leaves that turned yellow were those which had been punctured in the base of the petiole or in which the punctures in the stem were so close to the petioles that the necrotic lesions about the punctures might easily have affected the leaves. These inoculated potato plants continued to grow and showed no further symptoms of disease. Their progeny are now being grown and appear to be healthy. None of the check plants and none of the plants inoculated with other viruses showed these local necrotic lesions at the points of inoculation.

The results in this test are in accord with those reported by Johnson (6). This suggested at once that the variety of potatoes used is an important factor in the results, for Johnson apparently used only Bliss Triumph potatoes for his experiments with tobacco mosaic on potatoes. The results also suggest that in this variety the rapid killing of the tissues about the

point of inoculation with tobacco mosaic virus may protect the plant in some way from becoming systemically invaded.

On April 7, when it had already become evident that different results were being secured on Bliss Triumphs than had previously been obtained on Green Mountains, inoculation experiments were extended to include No. 9 potatoes (a variety belonging to the Rural group). Eight plants of this variety inoculated, as previously described, with tobacco mosaic virus did not develop necrotic lesions at the points of inoculation, although some of the lower leaves turned yellow and dropped. Necrotic lesions developed at the tips of a few leaflets, and by May 4 such lesions had developed in the pith and nodes of the upper parts of the stem. On the new leaves of three of the plants there was a striking mosaic mottling. Later, mottling appeared on all plants. This mottling differed from types seen previously by the writer on potatoes, and resembled more nearly the usual mottling of mosaic on tobacco. The spots were rather large, sometimes involving half a leaf. The degree of malformation corresponded with the size of the spots. The color contrast between the light green and dark green areas was marked. These plants were in a high temperature greenhouse until symptoms began to appear. Some of them were then moved to a cooler house, but even there low temperatures could not be maintained at that time of year. Eight punctured check plants remained healthy and normal.

Some of the inoculated No. 9 potato plants were cut back and transferred to the field. The new leaves on side branches which had grown after the time of inoculation were later used as a source of inoculum for tobacco in the field. The inoculations made on tobacco in the field (July 22) were as follows: Five tobacco plants inoculated with tobacco mosaic virus from tobacco all became typically affected with tobacco mosaic (Aug. 3); ten tobacco plants inoculated from No. 9 potato affected with tobacco mosaic all became typically affected with tobacco mosaic (Aug. 3), and symptoms were not distinguishable from those on plants inoculated from tobacco. Of ten check plants alternating with the ten plants just mentioned, one became affected with tobacco mosaic, and nine remained healthy. Ten tobacco plants were also inoculated from healthy No. 9 potatoes and ten more from healthy Green Mountains, with numerous additional uninoculated checks. Under field conditions, no symptoms could be detected with certainty on these plants. Certainly nothing developed in any way resembling the tobacco mosaic referred to above. As a rule, such inoculations made from healthy potatoes to tobacco under greenhouse conditions have given a mosaic on tobacco with symptoms of Johnson's "mottle" type, which is readily distinguishable from tobacco mosaic.

At the time the inoculations of tobacco were made in the field, a number of potatoes were also inoculated. Of ten tuber units of No. 9 potatoes, one

stalk in each of two plants in each unit was inoculated with tobacco mosaic virus from tobacco. One half of the potato plants was inoculated with material from the same tobacco plant which furnished inoculum for the Bliss Triumph earlier in the season. The other half was inoculated with material from young tobacco plants on which symptoms were just beginning to appear after inoculation with tobacco mosaic from the bottle of old extract.

Parallel inoculations were made at the same time on an equal number of Green Mountain potatoes. These, however, were not tuber units but the ordinary run of seed from a relatively disease-free stock. Pairs of inoculated plants alternated in the row with pairs of check plants.

The inoculated Green Mountain potatoes developed symptoms a few days earlier than the Rurals and were showing rather marked streaking and spotting of stems and leaves during the week of August 13 to 20. These stalks were so severely injured that they made little or no further growth and died early in most cases, while the check plants and the uninoculated stalks in the same hills continued to grow vigorously.

All of the No. 9 potatoes inoculated with tobacco mosaic virus showed symptoms similar to those previously obtained in the greenhouse. In comparison with the checks they were considerably dwarfed. They continued, however, to make some new growth, and on this the leaves were mottled. The checks remained healthy with the exception of one unit which seemed to be affected with leaf roll, probably tuber transmitted.

Tuber progeny of some of the No. 9 and Green Mountain potatoes inoculated with tobacco mosaic virus in the field were grown in the greenhouse during the following winter. Under greenhouse conditions it soon appeared that temperature has a very important influence on the type of symptoms developed. Both No. 9 and Green Mountain potatoes grown at 15° to 18° C. appeared quite normal in the early stages of growth. Later a rolling of the leaves gradually developed, with a slight crinkling of the leaf surface. No mottling or necrosis was observed at this temperature. At a temperature ranging from 26° to 30° C., on the other hand, the two varieties behaved differently. The No. 9 potatoes, in addition to a general dwarfing, developed a very striking mottling of the leaves. The Green Mountain potatoes developed a necrotic streaking of the stems and spotting of the leaves as previously described. This occurred both in cases when potatoes were planted and kept in a warm house continuously and when they were brought into a warm house after having been started in a cool house. In the latter case the No. 9 potatoes developed mottling only on the leaves that grew after the potatoes were removed to the warm house. At the same time it was found that when inoculations were made with tobacco mosaic virus on potatoes of several varieties in the cool house, symp-

toms did not develop until they were removed to the high temperature house. Thus, while no systematic study has been made of the effect of temperature on the tobacco mosaic disease on potatoes, enough has been done to indicate clearly that it is a high temperature disease—which seems to distinguish it from the other mosaics of potatoes, at least from those whose temperature relations have been described. Yellow dwarf, which is also a high temperature disease, has quite different symptoms.

Inoculations were made on tobacco in the greenhouse from the progeny of some of the potatoes that had been inoculated with tobacco mosaic virus. The following are typical of the results secured. Five young tobacco plants inoculated with tobacco mosaic virus from tobacco were affected with typical tobacco mosaic. Five tobacco plants inoculated from No. 9 potatoes, progeny of potatoes inoculated with tobacco mosaic virus in the field, were affected with a combination mosaic as described by Johnson, characterized by early appearance of symptoms and considerable necrotic leaf spotting in addition to mottling. Five tobacco plants inoculated from No. 9 potatoes, progeny of potatoes inoculated with tobacco mosaic virus in the greenhouse, were affected with the combination mosaic. Five tobacco plants inoculated from Bliss Triumph, progeny of potatoes inoculated with tobacco mosaic virus in the greenhouse, were affected with a faint mottling of the larger leaves (described by Johnson as "mottle"), like that obtained on tobacco most commonly when it is inoculated from apparently healthy commercial potatoes. Ten uninoculated tobacco plants remained healthy. From these results it appears that the tobacco mosaic virus is transmitted by the tubers of No. 9 potatoes but not through those of Bliss Triumph.

In addition to results above reported, some preliminary observations were made on the effect of tobacco mosaic on other varieties of potato. Ten plants of each variety were inoculated, and ten kept as checks. The progeny of these potatoes have not yet been grown, and the results were not so clear cut as might be desired. Five plants of each variety were inoculated in the cool house (65° F.), but after about a month, as no symptoms were evident, they were removed to the warm house. In addition, another five plants of each variety were inoculated in the warm house. The inoculated plants of the Spaulding Rose variety were dwarfed, and necrotic lesions developed on the stems at the points of inoculation. The tops died irregularly, but the stems in many cases sent out new sprouts near the surface of the soil which were nearly normal in appearance, although there was a faint mottling and crinkling in some cases. The inoculated Early Ohio potato plants died quickly, and there was necrotic streaking of stems and spotting of leaves. In two cases new shoots developed which for a short time showed striking mottling, comparable with that developed on No. 9 potatoes, but these shortly

developed necrotic streaking and spotting also. Cobblers inoculated with tobacco mosaic virus all died at somewhat irregular intervals. Death followed the development of extensive necrotic areas in the stems. No mottling or spotting of the leaves was noted. Most of the Early Bovee potatoes inoculated with tobacco mosaic virus were alive after five months although somewhat dwarfed compared with the check plants. They also gradually developed a rolling of the leaves. The new leaves, of a few new shoots, were mottled and within a short time the tissue in the light areas died.

So far this tobacco mosaic disease has not been definitely connected with any disease occurring in commercial fields of potatoes. The symptoms on Green Mountains, however, so closely resemble those of the streak disease as to suggest at once that some part of this disease may be tobacco mosaic. The fact that the symptoms of tobacco mosaic vary so greatly on different varieties of potatoes and vary also under different temperature conditions makes possible a wide variety of symptoms under varying field conditions. Such a symptom complex has recently been described for the streak disease of potatoes in Europe by Atanasoff (2) under the name stipple-streak. However, as most of the potato varieties used are different from ours and no reference was made to the temperature conditions under which the work was done, it is not possible to say whether this is tobacco mosaic until further comparisons can be made. The marked effect of temperature, the successful infection of tobacco, the diverse symptoms on the three principal varieties of potato seem to offer good means of identifying this disease.

SUMMARY

The author's results agree with those obtained by Johnson and with those obtained by Fernow. The seeming difference arose from the fact that Johnson had used Bliss Triumph potatoes for inoculations with tobacco mosaic, while Fernow had used Green Mountains.

Tobacco mosaic produced different symptoms on different varieties of potato.

On Bliss Triumphs, local necrotic lesions were produced at the points of inoculation, with no systemic infection.

On Green Mountains the symptoms were much like those described for streak, consisting of streaks on the stems and veins and necrotic spotting of the leaves. This is usually accompanied in the second generation by extreme dwarfing and dropping of leaves. This raises the question as to whether some part of the disease diagnosed as streak in potatoes may not be tobacco mosaic.

On No. 9 potatoes, in addition to a limited amount of necrotic streaking and spotting, a mottling developed which resembled that of tobacco mosaic

on tobacco rather than the usual types observed on potatoes. They were also slightly stunted.

Preliminary experiments on other varieties of potato indicate that the tobacco mosaic virus causes a wide range of symptoms.

Marked symptoms were obtained only at relatively high temperatures, about 26° C. and above. No symptoms could be detected in young plants at temperatures from 15° to 18° C.

Tobacco mosaic virus is readily transmitted from potatoes back to tobacco, although in doing this one obtains not the tobacco mosaic disease alone but a combination disease consisting of tobacco mosaic and the virus disease commonly carried by apparently healthy commercial potatoes.

The needle puncture method, when rather numerous punctures are made, is effective for inoculating the tobacco mosaic virus into potatoes.

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"TARGET CANKER" OF APPLES AND PEARS

JOHN W. ROBERTS

The canker of apples and pears herein described under the name "target canker" is not a new one, but because it seems not to be generally recognized and because of its unusual prevalence in recent years it appears worthy of some attention.

The disease first came to the writer's attention in 1922 when he noticed two apple trees at Arlington Farm, Virginia, the trunk and older branches of which were almost completely covered with bark cankers. On both trees these cankers consisted of circular dead areas of bark in which there were cracks arranged in concentric circles. The concentricity of these circles was so nearly perfect that it suggested the name "target canker." In August of that year, on the current year's growth of water-sprouts, young cankers were noted which, starting as dark pimples, soon showed a characteristic arrangement of concentric circular ridges which later cracked open (Fig. 1).

The cankers are confined to the cortical layers and become more superficial with age, since they are cut out by the formation of cork underneath. Even cankers of the current year's growth collected in July have been found to be isolated by a layer of cork. On older twigs the cankers appear to start as raised pimples resembling those of measles.[1] Mr. H. B. Derr, county agent of Fairfax County, Virginia, states that in an orchard of young Delicious trees the disease is mostly on the northwest side of the trees. The writer has also noted that this is sometimes the case. Some trees appear to be seriously devitalized by the canker, but it is uncertain whether such trees owe their condition to the disease or whether their lack of vigor was a predisposing factor in its development.

At Arlington Farm two French seedlings about 15 years old were the most severely affected, there being scarcely a square inch of the trunks and branches free from the canker. Two Jonathan trees about 20 years old are badly affected at the present time. Three of six 8-year-old Delicious trees are affected, and one 16-year-old Grimes, now removed, was slightly affected.

The writer has received specimens of apple twigs and branches affected with the disease from Kentucky (Valleau, 1923), Virginia (Derr, 1927), and West Virginia (1927). Of commercial varieties, Delicious and Jona-

[1] Rhodes, A. S. Apple measles with special reference to the comparative susceptibility and resistance of apple varieties to this disease in Missouri. Phytopath. 14: 289–314. 1924.

than seem to be most susceptible. The writer knows of only two collections of the disease on pear, one from Georgia on the Kieffer variety (Scott, 1908) and one from California (Shear, 1924).

The cause of target canker is not known to the writer, but there are reasons for suspecting that it may be of non-parasitic origin. It is quite possible that it is of the same nature as measles, which it resembles in its early stages. Target canker and measles may be different manifestations of the same disease. The early stages of brown bark spot,[2] the cause of which is unknown, also resemble those of target canker.

A total of 96 cultures have been made from young cankers at various times. Of these, 54 have been sterile, various species of fungi have developed on 40, and bacteria have developed on 2. The fungi have been chiefly *Physalospora malorum* and species of *Coniothyrium* and *Alternaria*. No inoculations have been made, but inoculation work and further cultural work are contemplated. No fungi have been found fruiting on the cankers, and no mycelium has been found in sections of young cankers.

There is of course a possibility that insect punctures may be responsible, but the following observations made at Arlington Farm incline the writer to the belief that certain conditions of the environment, at present unknown, cause, or at least favor, the development of the disease.

The French seedling apples that were so completely covered with cankers had never been vigorous and were quite evidently growing under conditions unfavorable for their normal development.

The diseased Jonathan apples were affected with crown-gall at the time they were planted and are extremely small and feeble for their age. Other and more vigorous Jonathan trees of the same age are free or nearly free from the disease.

In a small 8-year-old orchard planted on a slope extending downward from east to west about 120 feet there are six trees of Delicious, two of which are near the upper part of the slope, two about half way up, and two at the bottom of the slope. The last two are much larger than the others, supposedly because of a more bountiful supply of water and nutrient materials. Neither of these trees has more than a slight trace of the disease. Of the other four trees, one is nearly free from the disease but the others have hundreds of cankers. In the writer's notes these diseased trees are referred to as Delicious trees 1, 3, and 4, respectively, and they will be so designated here. The following observations were made on April 14, 1927:

Tree 1. The affected branches are mostly on the west side of the tree, although the branches on the north side also have a few cankers. Certain branches, badly cankered throughout, arise from limbs which otherwise are

[2] Swingle, D. B., and H. E. Morris. The brown bark spot of fruit trees. Mont. Agr. Exp. Sta. Bul. 146. 1921.

FIG. 1. Target canker of apple. Above, on a three-year old, below, on a six-year old branch of a French seedling. Arlington Farm, Virginia. September 29, 1923.

not affected, the cankers ceasing abruptly at the point where the healthy and diseased branches unite. The tree grew very little last year.

Tree 3. Every limb has some target cankers. The branches on the west side are the most severely affected, those on the north are less affected, those on the east have only a few cankers, while the large and vigorous southernmost limb is almost free. One small twig of this limb is badly affected, the cankers stopping abruptly where the twig joins a larger branch. A small badly cankered branch on the northeast side of the tree is dead. The tree as a whole grew only slightly last year.

Tree 4. Branches on the west and north sides are badly cankered; those on the east and south sides show only occasional cankers. One branch which is near the center of the tree and which forms a fork with the most severely cankered branch has the least cankers of any of the larger branches. Here again the disease ends abruptly at the juncture of the two branches. The tree grew somewhat more vigorously last year than did tree 3.

The relatively small number of cankers on the more vigorous of the Delicious and Jonathan trees, the greater prevalence of cankers on the west and north sides of the trees, the frequent restriction of the cankered areas to certain limbs—which may be almost completely covered by them, and the apparent internal origin of the cankers indicate that the disease is of non-parasitic nature. These observations considered separately lend but slight support to this view, but taken together and considered with the fact that as yet no organism has been consistently isolated from the young cankers they make this view tenable.

As the writer has no experimental data on the subject, he is not able to make any definite recommendations for control. The observation that the more rapidly growing trees are relatively free from the disease suggests that if trees are kept in good growing condition they are not liable to be affected and that the recovery of affected trees may be hastened by employing the usual means for increasing their vigor.

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STUDIES ON THE CONTROL OF MILLET SMUT[1]

L. E. MELCHERS[2]

Although millet (*Chaetochloa italica*) is not a crop of major importance in Kansas, its value ranges between half and three-quarters of a million dollars annually. It is grown in a large number of counties in the state practically every year. The most important disease attacking this crop is the smut caused by *Ustilago crameri* Koern. In 1924 an unusual number of inquiries were made concerning millet smut control. The only known seed treatment at that time was the long-time formaldehyde treatment which required soaking the seed for two hours in a formaldehyde solution. This causes swelling of the seed and necessitates drying before it can be sown. The formaldehyde treatment, although effective, is not wholly satisfactory. In many instances farmers will sow smutted seed rather than treat it with formaldehyde.

Extensive investigations were being conducted on sorghum smut control at the Kansas Agricultural Experiment Station during 1925 and 1926. It seemed desirable to use some of the more promising fungicides on smutted millet seed and determine their effects on the viability of the seed and on smut. Both liquid and dust treatments were used. Table 1 gives the results obtained during 1925 and 1926. In 1925 sowings were made on two separate dates, while in 1926 only one sowing was made. All the seed was artificially smutted, the spore load being much heavier than ordinarily would occur on millet seed. It was desirable to ascertain the limits of efficiency of the several fungicides. Heavy infection was secured in the untreated checks.

The results of two years' data on millet smut control show that:

1. The wet treatments which are most effective for smut control are those with formaldehyde, copper sulphate, and Uspulun.

2. Formaldehyde, either dry or as a mist, should never be used for millet smut control. It seriously injures the germination of the seed.

[1] Contribution No. 269 from the Department of Botany and Plant Pathology. Cooperative investigations between the Kansas Agricultural Experiment Station and the Office of Cereal Crops and Diseases, Bureau of Plant Industry, United States Department of Agriculture.

[2] Acknowledgment: the author is indebted to C. O. Johnston and C. H. Ficke for their aid in these studies.

TABLE 1.—*Results of seed treatments for the control of millet smut, Ustilago crameri, at Manhattan, Kansas, 1925 and 1926*

Row no.	Treatment	1925						1926		
		Sown April 22			Sown May 11			Sown May 14		
		Total no. heads[a]	No. smutted heads	Percentage smut	Total no. heads[a]	No. smutted heads	Percentage smut	Total no. heads	No. smutted heads	Percentage smut
1	Formaldehyde 1–320, soak 1 hr.	.....	0	0.0	.....	0	0.0	152	0	0.0
2	do	.....	0	0.0	.....	0	0.0	126	0	0.0
3	Formaldehyde 1–320, soak 2 hrs.	.....	0	0.0	.....	0	0.0	147	0	0.0
4	do	128	5	3.9	.....	0	0.0	139	0	0.0
5	Check, smutted not treated	135	46	34.0	119	87	74.3	127	41	32.2
6	Dry formaldehyde, 3 cc. per qt. seed, covered 5 hrs.	no stand	....	.....	no stand	....	.....	65	0	0.0
7	Uspulun 1–400, soak 1 hr.	161	2	1.2	.....	0	0.0	117	1	0.8
8	Semesan 1–400, soak 30 min.	150	75	49.3	123	50	40.6	147	6	4.0
9	Germisan 1–400, soak 30 min.	134	5	3.7	107	11	10.3	169	3	1.7
10	Check, smutted not treated	111	46	41.4	141	59	41.8	107	63	58.8
11	Copper sulphate, 1 lb. to 10 gal. (1–80), soak 10 min.	.....	0	0.0	141	4	2.8	174	0	0.0
12	Super Kalimat 1–400, soak 15 min.	149	18	12.3	80	3	3.7	102	0	0.0
13	Copper carbonate (Cal. Chem. Co.), 2 oz. per bu.	111	6	5.4	.....	0	0.0	112	1	0.8
14	Copper carbonate (Cal. Chem. Co.), 4 oz. per bu.	.....	0	0.0	.....	0	0.0	119	0	0.0
15	Check, smutted not treated	115	28	24.3	99	42	42.4	131	57	43.5
16	Copper carbonate (Cal. Chem. Co.), 6 oz. per bu.	122	3	2.4	.....	0	0.0	104	0	0.0
17	Coppercarb, 2 oz. per bu.	139	3	2.1	125	2	1.6	78	1	1.2
18	do 4 oz. per bu.	.....	0	0.0	129	3	2.3	109	0	0.0
19	do 6 oz. per bu.	.....	0	0.0	157	2	1.2	87	1	1.1
20	Check, smutted not treated	104	28	26.9	126	54	42.8	109	47	43.1
21	Niagara Dusting Sulphur, 2 oz. per bu.	128	25	19.5	104	12	11.5	103	9	8.7
22	do 4 oz. per bu.	119	22	18.4	130	14	10.7	103	3	2.9
23	do 6 oz. per bu.	109	6	5.5	74	12	16.2	105	4	3.8
24	Bayer dust, 2 oz. per bu.	138	8	5.8	112	13	11.6	124	17	13.7
25	Check, smutted not treated	99	23	23.2	35	14	40.0	97	46	47.4
26	Sulphodust, 2 oz. per bu.	109	10	9.1	Row killed by chinchbugs			115	21	18.2
27	Colloidal copper, 2 oz. per bu.	50	4	8.0	do			109	3	2.7

[a] No count was made in rows where smut was entirely absent. The number of heads per row was approximately 110 to 130.

3. The most effective and economical dust is copper carbonate. At least 4 ounces of this dust per bushel of seed are required for the best results.

4. Other dusts, such as various sulphur dusts, Bayer dust, and colloidal copper dust, are not satisfactory for controlling millet smut.

These studies show that the laborious formaldehyde method for millet smut control may be replaced by the more convenient copper carbonate dust method. Not only is copper carbonate effective for the control of sorghum and millet smuts, but it is cheaper and easier to apply than formaldehyde.

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PHYTOPATHOLOGICAL NOTES

Increasing Prevalence of Hypochnus Rot of Apples.—In 1903, Eustace[1] described a rot of apples caused by *Hypochnus* sp. This rot was not reported outside of New York, and there only on Baldwin and Rhode Island Greening apples, where it was consistently associated with and apparently followed scab.

Eustace pointed out the similarities of the macroscopic appearance of Hypochnus rot and the pink mold rot caused by *Cephalothecium roseum* and described the differences as follows: "On fruit affected with *Cephalothecium roseum* there is usually a conspicuous white or pinkish growth of the fungus in the center of an affected spot; whereas the new fungus does not show at all conspicuously on the surface of a decayed spot until made to do so by artificial conditions. On fruit, *Cephalothecium roseum* is a very shallow growing fungus, penetrating the tissue not much more than an eighth of an inch, while the new fungus grows much deeper and in its late stage extends to the core." Eustace found that the *Hypochnus* fruited rarely in culture or on apples and indicated the diagnostic value of the clamp connections consistently present in the mycelium.

For the past two years a rot agreeing in essentials with that described by Eustace has been found on Baldwin apples on many of the eastern markets. Specimens have been found at Chicago, Milwaukee, New York, Cleveland, Detroit, Columbus, and Indianapolis. The rot is quite common on New York Baldwins, and it is noteworthy that, while cultures have been made from many of the specimens found, *Cephalothecium roseum* was not isolated in a single instance. This indicates that, at least during the past two years, Hypochnus rot has been much more prevalent on the markets than pink mold rot. Hypochnus rot has also been found on New York Greenings but less frequently than on Baldwins.

A fungus which has characteristic clamp connections and appears to be *Hypochnus* has recently been isolated from several lots of Winesaps from Yakima and Wenatchee, one lot of Winesaps from Southern Illinois, from Ben Davis and Roxbury Russett apples from New York, from Ben Davis apples from Southern Illinois, and from one lot of Jonathan apples from Idaho.

On Winesaps and Jonathans the lesion looks much like Northwestern anthracnose, in many instances showing a conspicuously tan center with a

[1] Eustace, H. J. Two decays of stored apples. N. Y. (Geneva) Agr. Exp. Sta. Bul. 235. 1903.

brown to black border. The necrotic tissue, however, is somewhat tough and stringy, in contrast to the soft consistency of the true anthracnose or bull's-eye rot lesions as found on the market. On Winesaps in storage the rot is not much in evidence until rather late in the storage season, and has not been found following scab. On Baldwin, Ben Davis, and Rhode Island Greening apples the decay is similar to that on Winesap and Jonathan but is most often found following scab. When infected apples are kept in a moist chamber, the mycelium spreads over the surface of the apples, radiating uniformly from the lesions and forming a characteristic, fine, closely appressed, white mycelial mat.—L. F. BUTLER, Office of Fruit Disease Investigations, Bureau of Plant Industry, Washington, D. C.

Purification of the Virus of Tomato Mosaic.—As a preliminary step in the study of the properties of the virus of tomato mosaic it has seemed highly desirable to free the virus as completely as possible from the various constituents of the plant juice and to obtain the virus in large quantities in a clear and colorless water suspension. This has been accomplished by two methods.

In one method the juice from crushed mosaic plants was filtered through a sintered glass filter and the residue on the filter repeatedly washed with distilled water until the filtrate was colorless. The residue was then suspended in distilled water, and the clear filtrate obtained was proved to contain the virus because young plants inoculated in an insect-free greenhouse became infected.

In the second and more effective method the juice from crushed plants was centrifuged at 35,000 revolutions per minute and the fairly clear supernatant liquid passed through a powdered charcoal filter. The powdered charcoal was then washed with distilled water until the filtrates were clear and colorless, after which the virus was liberated from the charcoal and obtained in a clear and colorless water suspension, which was shown by inoculations to be infectious.—P. H. BREWER, H. R. KRAYBILL, and MAX W. GARDNER, Purdue University, Lafayette, Indiana.

REPORT OF THE ELEVENTH ANNUAL MEETING OF THE PACIFIC DIVISION OF THE AMERICAN PHYTOPATHOLOGICAL SOCIETY

The meeting was held in conjunction with the annual meeting of the Pacific Division of the American Association for the Advancement of Science at the University of Nevada at Reno, and was called to order by President W. S. Ballard on the afternoon of June 23, 1927.

Twelve members were present.

During the business session a motion was passed that the society recommend to the Committee on Nomenclature the changing of the name Western Yellow Tomato Blight to "Tomato Yellows."

OFFICERS OF THE SOCIETY

President ..... W. S. BALLARD
Vice-President ..... W. G. MILBRATH
Secretary-Treasurer ..... B. A. RUDOLPH
Councilor ..... W. T. HORNE

B. A. RUDOLPH, *Secretary-Treasurer*

ABSTRACTS

Notes on fruit decays of the feijoa (Feijoa sellowiana Berg).—W. T. HORNE.

The feijoa is an ornamental shrub which has fruit for home use and local markets. It possesses unique qualities, and its season (November) gives it an interesting place following the summer fruits.

Studies carried on principally with fruit from the Citrus Experiment Station, Riverside, were the basis for the following conclusions. Gray mold (*Botrytis cinerea* Pers.) appears to be the most active and common cause of spoilage. The decay produced is moderately tough and becomes somewhat dry; the fruit is rather quickly invaded and characteristic flavors destroyed. In cool dry air infected fruit merely withers.

Apple green mold, *Penicillium expansum* Lk., is the next most important agent of decay. The blue and green molds of citrus were not found occurring naturally on feijoa, and as a result of inoculation developed only poorly. Anthracnose or citrus wither tip fungus, *Colletotrichum gloeosporioides* Penz., was found in one case. Numerous other molds, including *Alternaria*, develop abundantly on over ripe fruit.

The treatment of lime-induced chlorosis in fruit trees.—J. P. BENNETT.

Effective treatment of chlorotic pear trees growing in a calcareous soil has been accomplished by placing solid ferric citrate in holes bored into the trees below the graft-union and below the ground level. Holes 1 centimeter in diameter and 3 to 5 centimeters in depth were bored at intervals of 8 to 10 centimeters around the main

root, one-half gram of the powdered salt placed in each hole, and the holes closed with grafting wax. The treatment was most effective when applied during the dormant season; during the active season, injury to the leaves frequently resulted. Complete greening of all leaves in the season following the treatment has occurred in 90 per cent of the 5,000 trees treated; incomplete greening in the remainder is attributed to poor distribution of the iron salt as a result of too small a dosage. The effect of the treatment has lasted for two years. The cost of treatment varied between two and ten cents per tree, according to size, in trees from three to thirty years old. Similar results have been obtained with chlorotic prune and walnut trees. Effective treatments have also been carried out in the same manner with ferrous tartrate, ferrous sulphate, ferric malate, and other soluble iron salts.

Celery experiments.—T. E. RAWLINS.

The addition of 2½ per cent soil from celery roots to fertile potting compost was found to treble the early growth of celery plants in such compost. Those plants to which no celery soil was added were stunted, chlorotic, and had necrotic leaf margins, while those to which celery soil was added had a very healthy appearance.

Further experiments to determine whether this increased growth is due to the celery mycorhiza are in progress.

Inoculation experiments with western yellow tomato blight in relation to environmental conditions.—MICHAEL SHAPOVALOV.

Experiments were carried on which prove conclusively that the curly top virus, when introduced in the tomato plant by means of viruliferous *Eutettix tenella* Baker, may under specific environmental conditions produce a pathological phenomenon indistinguishable from that known as western yellow blight of tomatoes. It was practically impossible to attain the complex of field symptoms under greenhouse conditions, particularly with caged plants, but perfect specimens of blight were produced with tomatoes grown outdoors unprotected or in light muslin cages.

Shading retards the progress of the disease and partly prevents it. Under heavy shading certain field characteristics of blight are lacking, while symptoms of curly top are more conspicuous. Light and humidity appear to be the most important single factors in the development of the complete field symptomology. High temperature may shorten the incubation period but in the absence of proper light and humidity conditions cannot bring about the characteristic blight complex. Certain other factors, such as the age of the plant, may either strengthen the resistance of the host or weaken it.

It is suggested that the name "western yellow tomato blight" be changed to "tomato yellows": (a) for the sake of brevity; (b) to conform with facts, and (c) to include similar diseases of other crops.

Relation of temperature to growth of Penicillium italicum and P. digitatum and to citrus fruit decay produced by these fungi.—H. S. FAWCETT and W. R. BARGER.

On culture media as well as on orange fruits, *P. italicum* and *P. digitatum* both appeared to have nearly the same optimum temperature for development. The difference, however, between the rate of growth at the optimum and the rates at higher and lower temperatures was greater in the case of *P. digitatum* than *P. italicum*.

In most cases the rate of development of decay was much more rapid at the stem end of the orange fruits than at the stylar end. The percentage of decay due to

injuries near the stem end was also greater in most cases than that due to equal injuries near the stylar end.

In lots of fruit inoculated with *P. italicum* and held respectively at 66.8, 74.8, and 80.6° F., nearly all fruits started to decay in 4 days, while lots held at 86 and 57.5° F. were in similar condition in 8 days, and lots held at 50° F. in similar condition in 12 days.

In lots inoculated with *P. digitatum*, much the same general relation of temperature to time and percentage of decay was noted, except that apparently the temperatures above and below the optimum had a greater relative inhibiting effect than in lots inoculated with *P. italicum*.

The enzymes of Pythiacystis citrophthora Sm. and Sm.—L. JOS. KLOTZ.

In a survey of the enzymes of the mycelium of *Pythiacystis citrophthora*, the following were tested for: esterases, cellulase, cytase, pectinase (pectase), inulase, diastase, raffinase, invertase, lactase, maltase, emulsin (amygdalase, salicinase, arbutinase), glucosidases that attack hesperidin and phloridzin, tannase, amidases (deaminases, deamidases, urease), histozyme, proteases, rennet, zymase, peroxidase, oxidase, catalase, reductase, and tyrosinase.

Very positive evidence was obtained for the presence of some of the lower esterases, for diastase, invertase, maltase, emulsin, phloridzinase, asparaginase, urease, peroxidase, and catalase; less evidence is forthcoming for the presence of cytase, lactase, hesperidinase; there was very slight indication of the presence of inulase, pectinase, protease and glycolase; and for the remaining enzymes sought the results were entirely negative.

The necessity for check determinations on both the active and deactivated enzyme material is emphasized, and a more accurate method of calculation given.

The diastase of this fungus attacks gelatinized starch vigorously, but starch suspended in cold water only feebly.

Although urea solution of the strength tried could not be used by the fungus, the enzyme powder gave a strong urease reaction.

Ecological studies of curly-top of sugar beets.—WALTER CARTER.

High light intensity, temperature, and evaporation appear to favor the development of severe curly-top symptoms. These same factors apparently affect the number of positive cases obtained.

Experiments on the control of the external environment were conducted, using various pigments. The transmission capacity of sprayed and unsprayed leaves was measured with a Kimball pyrheliometer. Significant differences in the yield of beets sprayed with pigments were obtained.

Dehulling barley seed with sulfuric acid to induce infection with covered smut (Ustilago hordei.)—FRED N. BRIGGS.

Experiments conducted by the writer in the greenhouse at Berkeley showed that barley seeds dehulled by sulfuric acid and inoculated with covered smut showed percentages of infected plants of the same magnitude as inoculated hand-dehulled seeds. The seeds were soaked in a quantity of acid from two to three times the volume of seed and then washed for 20 minutes under a faucet fitted with a shower or rose spray. Tennessee Winter barley grown from seed treated with various concentrations of sulfuric acid and seeded in the field in the fall of 1926 at Davis, California, showed the following percentages of infection with covered smut:

Concentration of acid	Duration of treatment	Plants			
		Produced		Smutted	
		Percentage	Number	Number	Percentage
Check	No treatment	80.0	60	5	8.3
do	Dehulled by hand	48.0	36	20	55.6
Conc.	5 min.	64.0	48	14	29.2
do	10 do	30.7	23	11	47.8
90 per cent	15 min.	18.7	14	9	64.3
do	20 do	6.7	5	1	20.0
80 per cent	25 min.	38.7	29	13	44.8
do	35 do	4.0	3	1	33.3
70 per cent	30 min.	69.3	52	11	21.2
do	40 do	74.7	56	29	51.8
do	50 do	45.3	34	23	67.6
60 per cent	1½ hrs.	69.3	52	22	42.3
do	2 do	48.0	36	19	52.8
do	2½ do	56.0	42	27	64.3
50 per cent	4 hrs.	36.0	27	14	51.9
do	5 do	24.0	18	14	77.8
do	6 do	16.0	12	9	75.0
40 per cent	6 hrs.	58.7	44	25	56.8
do	7 do	58.7	44	24	54.0
do	8 do	61.3	46	35	76.1
30 per cent	24 hrs.	17.3	13	9	69.2

(Cooperative investigations by the Office of Cereal Crops and Diseases, Bureau of Plant Industry, United States Department of Agriculture and the Agronomy Division of the California Agricultural Experiment Station).

A cytological study of Puccinia coronata Cda. on Banner and Cowra 35 oats.—MABEL L. RUTTLE, and W. P. FRASER.

The development of uredinia and telia of crown rust (*Puccinia coronata* Cda.) on Banner oats (susceptible) and Cowra 35 oats (variable in resistance) was studied cytologically. The formation of appressoria and entry take place in the same fashion on both. Appressoria are sometimes lobed. The substomatal vesicle is first a rounded sac which changes later into a canoe-shaped body from each end of which a slender infecting hypha is given off. The binucleate haustorium mother-cell is about 12 μ long. Haustoria on Banner are unbranched and uninucleate, attain a maximum length of 30 μ, function for a few days, are drained, and then appear as empty shells. Haustoria on Cowra 35 attain a maximum length of 18 μ, function briefly if at all, are seldom drained, and are frequently killed. In infected plants of Banner the host tissues give evidence of being stimulated by the fungus and show heightened turgor. The nuclei move over to the haustoria, increase somewhat in size, then contract and die. On Cowra 35, attacked cells often collapse and die, and adjoining cells show increased turgor, impoverishment, and enlargement of nuclei. Numerous yellowish-brown, rounded intracellular bodies varying in diameter from 3 to 18 μ are found in old infections and uninfected tissues dying of old age. Paraphyses are sometimes present at the margin of the uredinium.